

DISEASES OF THE
UPPER RESPIRATORY TRACT

THE NOSE, PHARYNX & LARYNX

P. WATSON WILLIAMS

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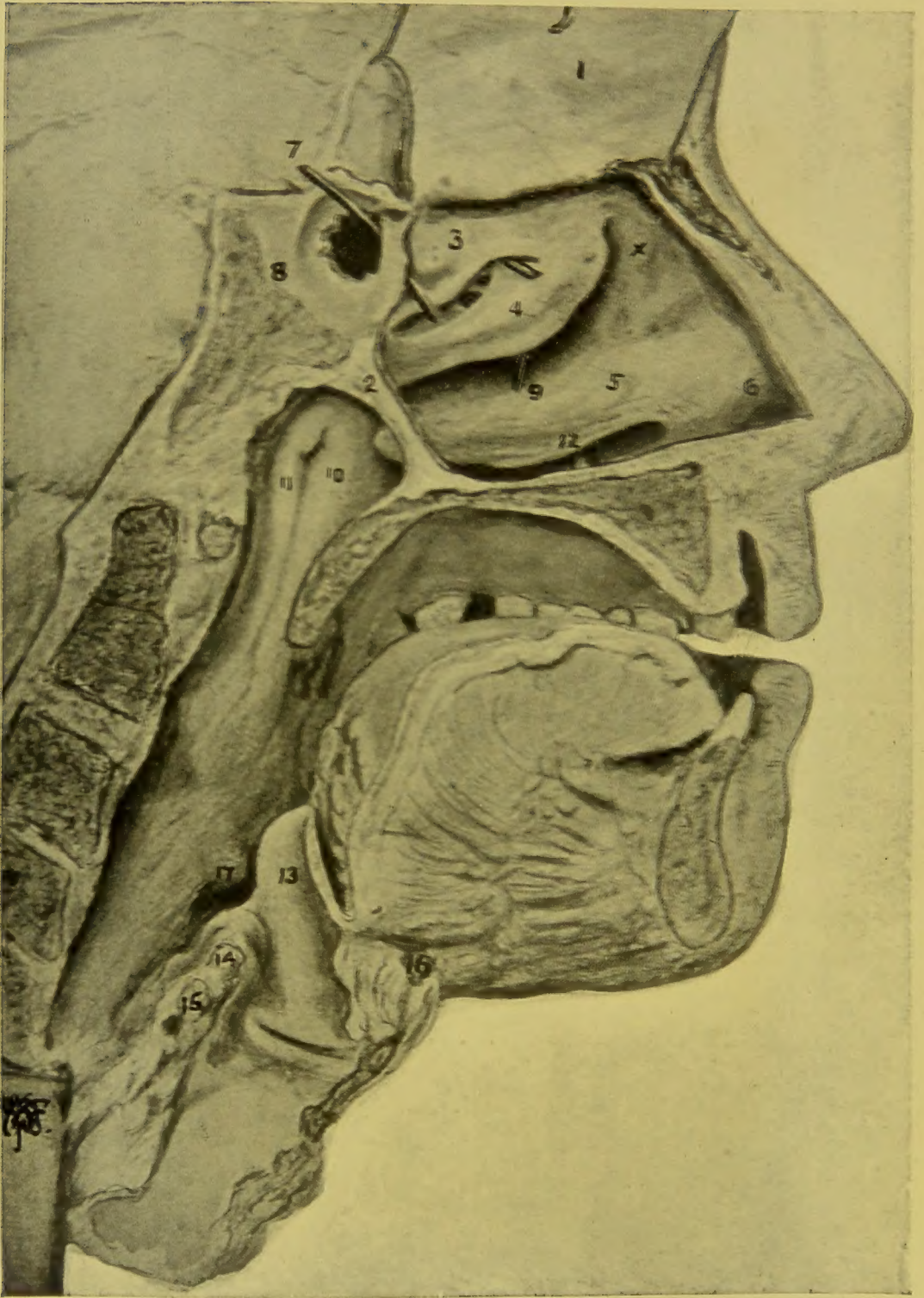
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DISEASES OF THE
UPPER RESPIRATORY TRACT.



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THE UPPER RESPIRATORY TRACT.

From a Male subject, reproduced five-sixths actual size ; being about the actual size in the Female.

THE UPPER RESPIRATORY TRACT.

A Dissection to show the anatomical relations of the interior of the nose, rhino-pharynx, pharynx, and larynx, and the various structures entering into their formation.

A vertical mesial section has been made, and the septum nasi (1) cut and raised to show the structures on the outer wall of the nose, the posterior margin of the septum nasi (2) being left *in situ* to show its relations to the Eustachian tube, etc.; (3) the superior, (4) the middle, (5) the inferior turbinated bodies, collapsed and shrunk, beneath which are respectively the superior, middle, and inferior meatuses. The left sphenoidal sinus has been broken into and a bristle passed through its aperture of communication with the superior meatus. More anteriorly, in the superior meatus, is seen a bristle passed into an opening into the posterior ethmoidal cells. Additional apertures are seen beneath this bristle; (6) the vestibulum nasi; (7) corresponds with the inter-peduncular space, the anterior cerebral artery appears immediately in front of the figure; (8) the body of the sphenoid; in the middle meatus is seen a bristle (9) passed upwards and forwards through the hiatus semilunaris and infundibulum, to the frontal sinus; (10) is placed on the salpingo-palatine fold, the orifice of the Eustachian tube is seen, and behind it (11) the salpingo-pharyngeal fold, the fossa of Rosenmüller being above and behind the posterior lip of the Eustachian; (12) a bristle passed into the lower end of the nasal duct; (13) the epiglottis; (14) the left arytenoid cartilage, just above it is the prominence of the cartilage of Sanatorini, and, more externally, of the cartilage of Wrisberg; (15) cricoid cartilage; (16) section of the body of the hyoid bone; (17) is on the prominence produced by its greater cornu; between 17 and the epiglottis is the fossa innominata, or pyriform fossa; the orifice of the left sacculus laryngis is seen bounded by the vocal cord below and the false cord above; (X) agger nasi, and below this eminence is the atrium meatus medii.

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DISEASES OF THE UPPER RESPIRATORY TRACT

THE NOSE, PHARYNX AND LARYNX.

BY

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TO
EDWARD LONG FOX, M.D. (OXON.), F.R.C.P.
AND MY COLLEAGUES
ON THE STAFF OF THE BRISTOL ROYAL INFIRMARY.

Preface to the Fourth Edition.

THE last edition of this Manual has long been sold out, the delay in the preparation of the present issue being due in part to the technical difficulties that had to be overcome in the reproduction of stereoscopic plates and in providing a suitable stereoscope which should be simple in use and always ready for reference to the plates.

The text has been revised throughout, largely re-written, and brought up to date, without departing from the original design, viz., a simple, concise, and thoroughly practical textbook on a scientific basis, affording information on every point likely to come within the needs of the practitioner and student of laryngology. Nevertheless, the size of the volume has considerably increased, mainly from amplification of the sections on "Diphtheria" and on "Diseases of the Nasal Accessory Sinuses," and to a very large increase in the number of illustrations. Some new sections have been added on subjects of clinical importance, all of which should make the book of greater service in actual practice. Yet by the introduction of smaller type the reader is enabled to disregard matters of detail, which would be of interest mainly to the advanced student or to the practitioner for reference.

It is often very difficult to obtain access to good anatomical preparations of special regions, and therefore I believe that the stereoscopic plates will be very helpful in affording realistic illustrations of the clinical anatomy of the nose, pharynx, larynx and ear, familiarity with which is absolutely essential for successful practice.

In addition to those I have already had occasion to thank, I must express my deep obligation to Sir Felix Semon for many hints and invaluable help in my work afforded during our recent collaboration in joint contributions to Prof. Allbutt's "System of Medicine"; to Drs. Gleitsmann and Chappell, of New York, Prof. Hamilton, and Drs. StClair

Thomson, Middlemass Hunt, Tilley, Walker Downie, besides many others, for kindly providing me with illustrations which depict various diseases in phases more typical than have occurred in my own practice, or, at least, in my collection of drawings; also to Dr. Dundas Grant, the late Prof. Kanthack, and other friends, for assistance in various directions.

Owing to the limitations of space imposed on me, it has been impossible to realise my wish to introduce a complete bibliography, or even to make all the acknowledgments that laryngology owes to many original workers. But I have been especially indebted to the "Centralblatt f. Laryngologie und Rhinologie," and to the "Journal of Laryngology, Rhinology and Otology," for many references and much aid.

Further, I would gratefully acknowledge the valuable co-operation of Drs. Geo. Parker and Kenneth Wills in the revision of proof sheets.

P. W. W.

1, VICTORIA SQUARE,

CLIFTON,

March, 1901.

From the Preface to the Third Edition.

THE fact that the First Edition of this work was sold out within a few months of its appearance, and that the Second Edition of a thousand copies has in the meantime become exhausted, is sufficient to indicate that it was needed.

Illustration is essential in describing affections the diagnosis of which largely depends on the appearances presented, and, with few exceptions, I have reproduced sketches from my clinical notes, believing that they will convey the most accurate idea of the aspects of disease, without elaboration.

I am under a sense of deep obligation to Dr. McBride, and Mr. Young J. Pentland, for permission to reproduce *Fig. 3, Plate IV*; to Professors Chiari, Emil Zuckerkandl, Dr. Riehl, of Vienna, and to M. Wagnier, of Lille, I am similarly indebted; while I beg also to thank Dr. W. P. Northup, of New York, Mr. Mark Hovell, the "Scientific Press," and Messrs. J. Wright & Co., for their assistance in preparing the work for the press.

P. W. W.

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ERRATA.

Page 3, line 10, for "ethnoidal" read "ethmoidal."

.. 15, .. 5 *from the bottom, for "instrinsic" read "intrinsic."*

.. 23, .. 4, *for "viz." read "and."*

.. 23, .. 6, .., *"palantine" read "palatine "*

.. 59, .. 28, .., *"Dalton's" read "Galton's."*

.. 60, .. 12, .., *"fenestrum ovale" read "fenestra ovalis."*

.. 61, *for "Rinné" read "Rinne."*

.. 61, *line 2 from the bottom, for " points to" read " Rinne + points to."*

.. 67, *line 5, for "three fourths" read "half."*

Plate xxxvii, Fig. 2 is the left side and Fig. 3 the right side.



DISEASES OF THE UPPER RESPIRATORY TRACT.

CHAPTER I.

ANATOMY AND PHYSIOLOGY.

IN a work addressed to medical practitioners and senior students it may be assumed that every reader is acquainted with the topography of the regions under discussion, and has at hand the usual text-books of anatomy and physiology; yet it is desirable to draw attention to points of special importance from a *clinical* aspect. Here and at the end of the book a number of stereoscopic plates are given, forming a series of illustrations which yield a view of the anatomical preparations precisely as though they were actually placed before the reader in the best position for observing the features to be noted. As the parts presented are described with each plate it is only necessary here to refer to them very briefly.

THE NOSE.

In *Plate I (frontispiece)*, from a dissection in the museum of the University College, Bristol, the anatomical relations of the whole upper respiratory tract are well shown.

The nasal cavity is divided into two parts by the *septum*. To show the structure of the nasal fossæ I have cut three sides of the septum, and raised it as a lid, and have passed bristles through the openings in the walls of the fossæ, by means of which the accessory sinuses communicate with the nose.

The plates, being exact reproductions from nature, will afford precise information as to the relations of the different regions of the nose, pharynx, and larynx; and will be found very useful as an aid in performing various manipulations necessary for the diagnosis and treatment of disease of the nose and throat.

Plate XXVI shows the osseous structures forming the outer wall of the nose; *Plate XXVII*, the outer wall, covered with mucous membrane: *Plate XXVIII*, a vertical transverse section, should also be turned to; it shows in section the position and shape of the turbinal bodies, and the narrow thin roof of the nasal cavity formed by the cribriform plate. The nasal cavity has an inner and outer wall, a floor and a roof.

The *roof* is highest in the centre where it is also narrowest, being formed by the cribriform plate, and only two or three millimetres wide. In front it is formed by the frontal and nasal bones, and behind by the body of the sphenoid. The *floor* varies in width from 12 to 15 mm. ($\frac{1}{2}$ to $\frac{5}{8}$ of an inch). The inner wall is formed by the septum.

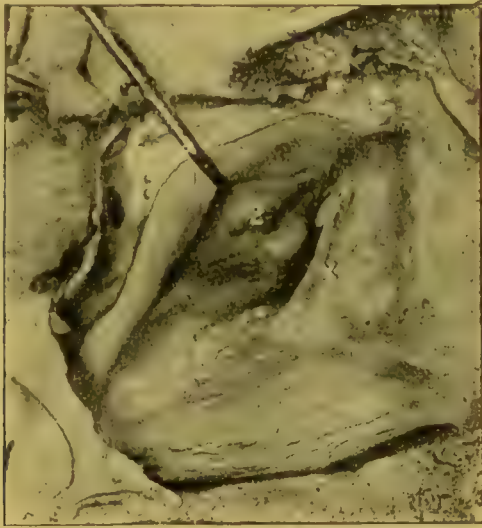


FIG. 1.

A part of the frontispiece dissection enlarged to the natural size. The middle turbinated body 1, has been raised by a hook, exposing (2) the middle meatus and showing the hiatus semilunaris immediately behind the processus unciniformis X.

In order to give a better idea of the position and size of the "hiatus semilunaris," or orifice by which the infundibulum, the anterior ethmoidal cells and the antrum of Highmore communicate with the middle meatus of the nose, I have drawn up the middle turbinated body by means of a hook, and have had these portions of the dissection reproduced in their natural size.

The outer wall is somewhat complicated, but is very important

from a clinical standpoint and therefore merits fuller consideration. It presents three transversely projecting shelf-like bodies, the superior, middle, and inferior turbinated bodies; the long irregular longitudinal passages beneath them being the superior, middle, and inferior meatuses, in each of which are the openings of the accessory nasal sinuses. A fourth turbinal body, the *concha superior*, or *concha Santoriniana*, is sometimes present above the superior turbinated body. Zuckerkandl found it in fifty-five out of one hundred and fifty specimens. The orifice of the sphenoidal sinus on each side is in the spheno-ethmoidal recess posteriorly, a few millimetres below the roof, and the openings of the posterior ethmoidal cells are seen in the superior meatus. In the middle meatus is a crescentic groove, the *hiatus semilunaris* into which the frontal sinus opens anteriorly; the orifices of the anterior ethmoidal cells are generally close to the infundibulum or frontal sinus opening. In the posterior portion of the hiatus semilunaris is the orifice of the maxillary sinus, the *ostium maxillare*. Sometimes the frontal sinus opens directly into the maxillary sinus, or both the frontal sinus and its duct may be absent. The anterior ethmoidal cells also sometimes open into the maxillary sinus. There may be a second opening to the maxillary sinus in the middle meatus, behind the hiatus semilunaris, the *ostium maxillare accessorium*; or, on the other hand, a maxillary opening directly into the nasal passage may not exist.

The lachrymal or nasal duct opens into the inferior meatus, the opening being generally as far back as the second bicuspid tooth. A comparison between the sizes of these apertures in the skeleton and when covered by mucous membrane, shows how greatly they are narrowed or even almost closed by the thick mucous membrane during life.

The *mucous membrane* of the nasal fossæ is called the pituitary (from the nature of its secretion) or Schneiderian membrane. It is continuous with the skin through the anterior nares, with the mucous membrane of the pharynx posteriorly, and with the lining of the accessory sinuses of the nose, also through the nasal duct with the conjunctiva, and posteriorly by the Eustachian tube with the middle ear. It contains very numerous mucous glands, but these are most abundant at the middle and back parts of the outer wall and septum. The physiological

functions of the nose are three-fold: vocal, olfactory, and respiratory, and the epithelium varies in character in different parts of the nasal passages, according to the different functions of these parts. Thus in the vestibulum near the orifice of the nose, where common sensation is required, squamous epithelium is found; in the olfactory region, where the terminal filaments from the olfactory bulb are distributed (see *Plate XXXIX*) the epithelium is columnar and non-ciliated and is pigmented, and lying beneath it are the olfactory cells of Max Schultze, but with these restrictions the whole of the nasal epithelium is ciliated.

The *arteries* of the nasal fossæ.—The most important is the sphenopalatine, from the internal maxillary which passes into the cavity of the nose, through the sphenopalatine foramen at the back part of the superior meatus. Here it divides into an internal branch to the septum, the nasopalatine, and external branches supplied to the lateral wall of the nose, the maxillary antrum, and the ethmoidal and sphenoidal sinuses. In passing from the sphenopalatine foramen to the septum the nasopalatine artery crosses in front of the body of the sphenoid only a few millimetres below the aperture of the sphenoidal sinus, and is therefore liable to be injured in trephining the sphenoidal sinuses. The other arteries are the alveolar branch of the internal maxillary to the antrum; the anterior and posterior ethmoidal to the roof of the nose, and the ethmoidal and frontal cells; and a twig of the small meningeal.

The *veins*, after forming a close network beneath the mucous membrane and in the turbinal bodies especially, pass out with the sphenopalatine and ethmoidal arteries, but some through the alveolar branch join the facial vein, and others, it is important to note, communicate with the veins in the interior of the skull through the cribriform plate and sometimes, especially in children, through the foramen cæcum. Such intercommunication between the veins (and accompanying lymphatics) of the nose and face and of the nose and cerebral sinuses, explains the clinical association of intra-nasal disease with facial erysipelas, etc., and cerebral symptoms respectively.

The *physiological functions* of the nose are, as already stated, threefold: olfactory, respiratory, and vocal. The first named is sufficiently noticed in other parts of the book.

The *respiratory* functions comprise : (1,) Warming, (2,) Moistening, and (3,) Purifying the inspired air.

While the superior half of the nasal fossæ is mainly concerned with the function of smelling, the mucous membrane of the middle and inferior turbinated bodies contains vascular erectile tissue, which warms and moistens the inspired air. In these regions it consists of connective tissue, the surface of which is covered with columnar ciliated epithelium, the deep portion forming the periosteum. Between these two layers are lymphoid tissue, an abundant supply of lymphatics, and numerous venous plexuses, into which the capillary vessels freely open, as shown in the accompanying illustration of a section of a hypertrophied inferior turbinal removed by snaring.

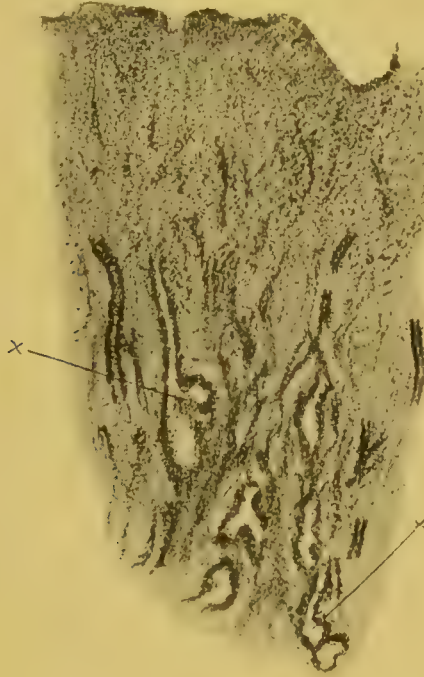


FIG. 2.

Section of hypertrophied erectile tissue from the inferior turbinated body : showing the arrangement of the vessels and venous spaces (x) into which the collecting veins are seen to open.

Around the venous plexuses unstriated muscular tissue is distributed. When the plexuses are filled the turbinal bodies swell considerably, but numerous elastic fibres in the deeper layers cause the tissue to collapse, unless actively distended by the peculiarly arranged vascular supply. Similar erectile tissue

is found occupying the lower part of the septum opposite the anterior extremity of the middle turbinal body.

We know that "mouth-breathing" is not only a source of inconvenience, but is generally attended with troublesome affections of the lower respiratory tract. The nose therefore performs important functions which cannot be fulfilled by the mouth, and a careful examination of its structure affords a ready explanation of these functions: for it can easily be conceived that these highly vascular mucous tissues are capable of rapidly warming inspired air as it passes over them, and of secreting with equal rapidity a copious supply of watery mucus which can yield its moisture to the warmed air. That this actually does take place has been demonstrated by Gréhant, Kayser, Bloch, MacDonald and others; and it has been proved by these observers that: (1,) Whatever the temperature of the atmosphere, the air, after ordinary inspiration through the normal nasal passages, is always raised to the temperature of the blood before reaching the pharynx; (2,) The air, after passing through the nose, is invariably completely saturated with moisture.

It has been estimated that about a half a pint of water is taken up by the inspired air in the course of twenty-four hours, and, if the air is to reach the lungs in the normal condition of saturation, it is obviously impossible for that amount of moisture to be taken up from the bronchial mucous membrane without serious risk to its physiological integrity. We know that we can breathe normally all the night through, and awake in the morning with the pharynx and larynx in a moist and healthy condition; but if we have lain an hour or two breathing through the open mouth, the pharynx is quite dry, or is covered with a tenacious thick mucus, and if this mouth-breathing becomes a habit, the pharynx and larynx suffer from chronic congestion, and the parts are "relaxed." It is, in fact, unnecessary to dilate on the pernicious effects of mouth-breathing; the constant tendency to laryngitis, attacks of bronchitis, and various other ways in which the evil effects are manifested, are familiar to practitioners. But a further function, scarcely less important than those already mentioned, is subserved by nasal respiration, viz., the purification of the inspired air; and this is worthy of careful consideration.

Bacteriology of the normal nasal passages.—It has been calcu-

lated by StClair Thomson and Hewlett that, at the lowest estimate, fifteen hundred organisms are inhaled into the nose every hour, while it must be a common event in the average London atmosphere for fourteen thousand to pass into the nose in a single hour. What becomes of them?

It has been shown by the researches of several observers that expired air is practically germ free, and that all or almost all micro-organisms have been arrested in the nasal passages before the inspired air reaches the trachea. It was therefore, but erroneously, assumed that the nasal passages must be crowded with micro-organisms; for the most part the micro-organisms and various impurities are arrested in the vestibule by the vibrissæ, and those that pass this natural barrier during inspiration are rapidly removed from the nasal mucous membrane, partly by the downflow of mucus and partly by the action of the ciliated epithelial covering.

Wurtz and Lermoyez found that as a rule the nasal mucus was sterile, and Thomson and Hewlett also made numerous cultures from the vestibule, the vibrissæ of the vestibule, and no less than seventy-six cultures from the interior of the nose; of the latter sixty-four remained absolutely sterile (84%). But numerous colonies were generally obtained by cultures from the vestibule and vibrissæ. Thomson and Hewlett do not affirm that micro-organisms are completely absent from the Schneiderian membrane, but that under normal conditions they are quite exceptional. They have further shown that if a culture be deposited on the septum the organisms are rapidly removed by the action of the ciliated epithelium, and that in two hours none of the myriads of organisms could be detected by their bacteriological cultures. They further show that while nasal mucus is not germicidal, it exerts an inhibitory influence on the growth of micro-organisms.

Park and Wright have re-investigated these questions, making cultures from the mucus in the interior of the nose of thirty-six normal individuals, with the result that only six were sterile and thirty non-sterile; but in most of the latter the colonies were not numerous, and though these results demonstrate that the nasal fossæ are not sterile, they are not so rich in microbes as was formerly supposed *à priori*. They attribute this to be due to the action of gravity causing a down-flow of mucus from the upper passages, to the action of cilia, to the fact that the mucus is not a good culture medium, to the filter action of the vibrissæ, and to the fact that inspired air usually contains few pathogenic micro-organisms. It is remarkable that many children with chronic membranous rhinitis which yields pure cultures of fully virulent diphtheria bacilli suffer from no constitutional symptoms whatever, while tubercle bacilli and various pyogenic cocci can rarely be found in the nasal passages of healthy individuals.

THE ACCESSORY SINUSES OF THE NOSE.

A fairly accurate knowledge of the anatomical conformation and relations of these accessory sinuses is essential in rhinology ; it will therefore be necessary to refer somewhat fully to the numerous features of clinical importance which they severally present.

THE MAXILLARY SINUS (ANTRUM OF HIGMORE).

To show the antrum of Higmore, the malar process has been removed from the skull in *Plates III* and *XVI*, and *Plates XVI* and *XXVIII* display the antrum in coronal sections.

The antrum is a three-sided pyramidal cavity hollowed out of the body of the superior maxillary bone, its base being formed by the outer wall of the nasal fossa and its apex by the malar process. The *roof* is formed by the floor of the orbit, the floor by the hard palate and the alveolar process, the anterior wall by the facial and the posterior wall by the zygomatic surface of the superior maxillary bone.

Reference has already been made to the *aperture* of communication with the nasal passage beneath the middle turbinated body. In a number of subjects, about 10 per cent. according to Zuckerkandl, there is an accessory ostium in the mucous membrane closing in the large bony aperture ; it is below the uncinatè process of the ethmoid, behind the ostium, and uncovered by the middle turbinated bone.

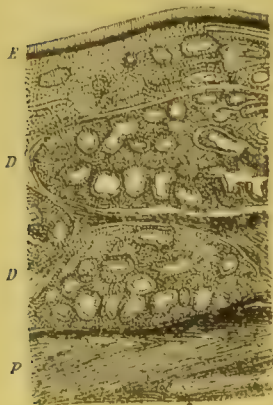


FIG. 3.

Section of the mucous membrane of the inner wall of the antrum, showing that the epithelium is ciliated, and that the membrane is rich in muciparous glands (ZUCKERKANDL).

The *floor* of the antrum is uneven from the projection of the sockets of the first and second molars, and several bony laminæ partially subdivide the cavity. These laminæ often interfere with drainage of pus from the antrum, and the position of the natural aperture shows that a collection of pus will escape more freely from the nose on lowering the head.

The relations of the posterior and anterior dental nerves, and of the superior maxillary nerve, which are well displayed in *Plate IV*, explain the cause of direct and reflex pains in antral disease. If tumours grow in the antral cavity or pus collects

PLATE II.

MESIAL SECTION OF A SKULL SHOWING THE RIGHT NASAL ACCESSORY SINUSES (natural size)



(1) Frontal sinus of the left side, separated by the oblique mesial septum from (2) the right frontal sinus; (3) Crista galli; (4) Cribriform plate; (5) Sella turcica (6) Optic foramen; 7, Sphenoidal sinus; (8) (9) Posterior ethmoidal cells; (11) Remains of the middle turbinal bone; (13) (14) Anterior ethmoidal cells; 15, Bulla ethmoidalis; 16) Processus unciniformis; 17 Opening into the maxillary antrum; 19) Remains of the inferior turbinal bone; (20) Position of the opening of the nasal duct in the inferior meatus; (22) Fronto-nasal duct; (23) Inferior meatus. The dotted line is drawn from the nasal orifice to the posterior wall of the sphenoidal sinus.

N B.—This preparation is displayed in stereoscopic relief in *Plate XXVI* at the end of the book.

and cannot escape, the walls of the antrum being thin readily yield, pushing up the floor of the orbit and displacing the eye, bulging into the nasal passage, or protruding outwards on the cheeks or backwards into the zygomatic fossa.

Development.—The maxillary sinus varies considerably in size, not only in different individuals, and at different ages, but even on the two sides in the same individual. In some persons the antra are very small, in others they extend beneath the nasal fossæ above the hard palate (see *Plate XVI*). It is formed earlier than any of the other accessory sinuses of the nose, its development commencing about the fourth month of foetal life. The antrum is small at birth and does not alter much in size until the second dentition, and then from about the sixth to the eighth year develops rapidly, but only attains its full dimensions in adult life.

THE FRONTAL SINUSES.

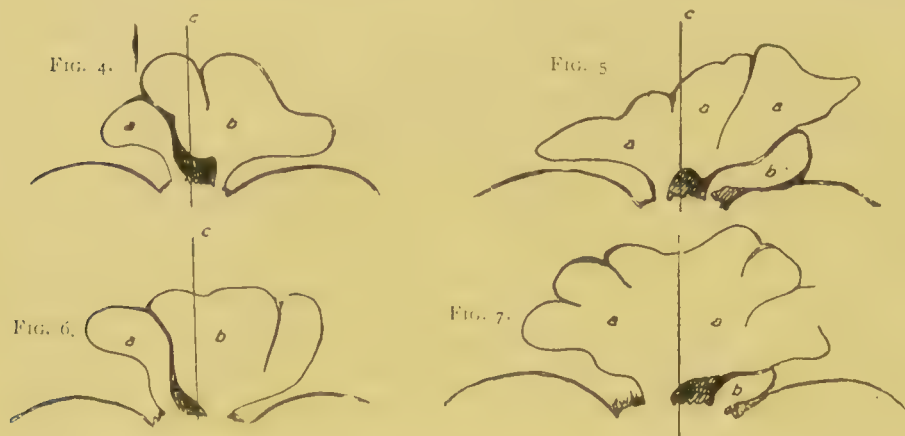
The frontal sinuses are two irregular cavities placed between the inner and outer tables of the skull in close proximity to the root of the nose, and are generally separated from one another by a thin bony septum. It will be seen in *Plate XX* that the separation of the inner and outer tables of the frontal bone forms an anterior and posterior wall, which, united above, are separated below by the floor of the sinuses, so as to form triangular-shaped cavities.

The *posterior wall*, dense but thin, is in relation with the frontal and olfactory lobes of the cerebrum. The sinuses extend horizontally backwards for a variable distance below the intracranial cavity and above the orbital fossa, sometimes reaching as far back as the ethmoidal notch.

The *anterior wall*, the thickest, usually extends downwards as far as the junction of the nasal bones with the frontal nasal process, but is variable in its upward extent. *Laterally* the frontal sinus in the adult generally extends to a short distance beyond the supra-orbital notch, but it may reach to the external angular process, and I have seen it extend backwards as far as the lesser wing of the sphenoid.

Logan Turner from an examination of 500 skulls found that in 357, or 71%, both sinuses were present; in 80, or 16·1%, both sinuses were absent; and in 63, or 12·5%, one sinus was absent. Tilley from an examination of 120 skulls, considers a sinus normal when it measures 28 millimetres from the median line outwards, *i.e.*, about as far as the inner third of the supra-orbital ridge; and normal in vertical extent when it measures 20 to 22 millimetres.

The *floor* is partly above the orbit, the orbital portion; and partly above the nasal fossa, the nasal portion in which is situated the ostium frontale leading to the naso-frontal duct which takes a direction downwards, inwards and backwards to reach the middle meatus of the nose. This communication between the frontal sinus and the nose is of great importance surgically, and will be described more fully below.



Outlines of frontal sinuses showing various degrees in irregular development and inequality between the two sinuses. In these figures the right frontal sinus is marked *a*, and the left one *b*. (TILLEY.)

The sinuses are almost always completely separated by a bony septum which is generally median below, but often deflected to one side or the other above. The septum may be either in a direct antero-posterior plane, or may be deflected so that one sinus comes to overlap the other, thus also making one sinus larger than the other; Gallemaerts found that the right sinus was usually larger than the left. Moreover, each sinus is often partially subdivided by one or more bony laminae which project from the floor and anterior wall, forming pockets in the floor, which interfere with drainage of the cavity. In a considerable number of cases one or more large anterior ethmoidal cells, or more correctly fronto-ethmoidal cells (Bryan), form a conical projection in the postero-internal angle of the sinus, the *bullæ frontalis*, and similar elevations may be produced by the upward projection of other anterior ethmoidal cells in the nasal portion of the floor, just external to the *ostium frontale*.

Development.—The frontal sinus has seldom appeared at all at birth, nor as a rule is it present before the seventh year. It is

usually well developed at puberty when the brain has arrived at its full size, but it is not generally fully developed before the twenty-first year. It is somewhat larger in men than women, but there is no certain means during life of determining the degree of development of these sinuses without trephining.

It owes its existence to the continuance of growth of the outer table of the skull, after cessation of growth of the inner table, which latter is always synchronous with the growth of the hair.

Even fairly well-marked superciliary ridges on a non-protruding supra-orbital area are frequently accompanied by poorly developed sinuses, or even absence of their vertical portion, and conversely Zuckerkandl found that large sinuses often exist with a flat supra-orbital region. But from an examination of 125 preparations, Lothrop considers that "in general it is fair to conclude that the more prominent the supra-orbital area, including both the superciliary ridge and the nasal eminence, the greater the probability of the presence of well defined sinuses."

Communication between the frontal sinuses and the nasal passage exists in all cases, usually through the infundibulum, but (owing to the variations in the arrangement and development of the frontal sinus and anterior ethmoidal cells described below), one finds here important anatomical differences.

Thus Lothrop in his 125 specimens found that (*a*,) in 53 per cent. there was no fronto-nasal canal, the frontal sinus opening directly into the "turbinate fossa" in the middle meatus. In a few cases the frontal sinus opened into the middle meatus through an irregular series of ethmoidal cells. (*b*,) An infundibulum was always present, the channel being partly bony, and a considerable portion membranous only. But in 53 per cent. of the specimens it had no connection with the frontal sinus, in these ending either (*a*,) in a cell of large size often corresponding to the agger nasi, or (*b*,) in one of the ethmoidal cells in the posterior angle of the frontal sinus (bulla frontalis), or (*c*,) in very few instances, the infundibulum was blind in its upper end.

Furthermore, he found that while it was difficult to pass a probe upwards from the nose to the frontal sinus, it could always be passed downwards, inwards, and backwards, and that it could not do any serious injury if the probe was forced through any obstructing cell in this direction. But in nearly 50 per cent. of cases a straight probe passed from the frontal sinus in this way entered the antrum of Highmore, and in a large number of the other specimens a slightly curved probe could be made to enter the antrum. The importance of this observation is obvious, accounting for the frequency with which pus escaping from the frontal sinus enters the antrum.

Irregularities. The frontal sinus may be very small or wholly absent. The bony septum may be incomplete and the two sinuses divided by a septum which is fibrous, or partly bony and partly fibrous, or again the

sinuses may communicate through the septum. In a small percentage of cases the frontal sinus is so elementary that it does not reach the supra-orbital margin. The sinuses are never bilaterally symmetrical, and on one side the sinus may be large and on the other very small. In the latter condition the large sinus may extend right across the median line.

THE ETHMOIDAL CELLS.

The ethmoidal cells (or ethmoidal labyrinth) occupy the whole of the lateral mass of the ethmoid bone, between the *os planum* or *lamina papyracea* externally and the *lamina cribrosa* or outer wall of the nasal passage, many of the cells being completed by articulation with the surrounding bones, *viz.*, frontal, sphenoid,



FIG. 2.

Diagram to show the arrangement of the accessory sinuses and their apertures of communication with the nasal passages. The dark oblique line indicates the attachment of the middle turbinate bone, dividing the various cavities into A, the anterior group, and P, the posterior group. The anterior group comprises F, the frontal sinus AE, the anterior ethmoidal cells, and the maxillary sinus, OM, being the ostium maxillare. The anterior ethmoidal cells are further divisible into (3) lower, AE', (2) middle, AE'', and (1) AE''', upper cells; I, the infundibulum. The posterior group comprises, PE, the posterior ethmoidal cells, and, S, the sphenoidal sinus.

and superior maxillary and lachrymal. The extreme thinness of the lamina papyracea or *os planum*, separating the ethmoid cells from the orbit is well shown in *Plate III*, defects in the bony lamina being not very rare, the orbit then being separated

by membrane only. The arrangement of the cells of the ethmoidal labyrinth is very irregular, but Zuckerkandl divides them into two groups: the anterior which opens into the middle meatus, and the posterior opening into the superior meatus, the transverse bony lamina which forms the line of demarcation between the two groups being fairly indicated by the incisura ethmoidalis inferior, or groove for the insertion of the middle turbinated body. The cells are separated by vertical septa (see *Plate III*).

The *anterior ethmoidal cells* from both the anatomical and clinical standpoint are closely associated with the frontal sinus. Hartmann finds that the frontal sinus is developed by the extension of an ascending recess (the *recessus frontalis*) in the anterior portion of the middle meatus, the cavity usually extending upwards between the plates of the frontal bones. By lateral excavations of the recessus frontalis the anterior ethmoidal cells are formed, and so these cells surround and open into the fronto-nasal canal. But the frontal sinus may be formed in another manner, *viz.*, by the pushing forward of one of these upper ethmoidal cells (which Hartmann terms frontal cells); or on the other hand the developmental process may stop short of the formation of a frontal sinus, or the frontal sinus may be present without any fronto-ethmoidal cells.

Other excavations developing in the lowest region of the recessus frontalis result in the formation of the other anterior ethmoidal cells at a lower level.

The *posterior ethmoidal cells* are a group of two or three large cells which open in the superior meatus close to the recessus spheno-ethmoidalis, or vertical groove between the ethmoid in front and the body of the sphenoid behind; their ostia are therefore just in front of the ostium sphenoidale.

The *bulla ethmoidalis* is the rounded eminence of an anterior ethmoidal cell lying beneath the anterior end of the middle turbinated body (see *Plate II*), and constitutes an important landmark.

It varies considerably in size, but Lothrop found it averaged 10 mm. in length, and projected 2 to 5 mm. into the middle meatus. It generally consists of one large cell (which may reach out to the orbital fossa), sometimes two super-imposed cells, with an oval ostium looking backwards from the upper part of its posterior wall, just beneath the turbinal.

The *bulla ethmoidalis* lies above the processus unciformis, forming the posterior boundary of the infundibulum and the upper border of the *hiatus semilunaris*. Ethmoidal cells may be present in the anterior angle of the middle turbinated body (the operculum of Schwalbe). Lothrop found these cells present in 18 per cent. of his specimens, some opening into the middle and some into the superior meatus. When the greater portion of the spongy bone is occupied by cells it is termed a *concha bullosa*.

Development.—The formation of the ethmoidal labyrinth does not commence till the fourth or fifth year, and is not complete till about the age of twenty.

These ethmoidal cells are very irregular in their development, and I have found that very often they are developed inversely in proportion to the size of the sphenoidal sinuses, in the specimens in which the sinuses generally were fairly developed. A single posterior ethmoidal cell may be as large as a cherry.

THE SPHENOIDAL SINUSES.

The sphenoidal sinuses, like the other accessory sinuses, are very irregularly developed. They are situated in the body of the sphenoid, and may vary in development from entire absence, to a cavity which occupies the whole of the body of the sphenoid and extends posteriorly almost as far as the foramen magnum. The two cavities are generally separated by a bony septum, but are rarely of equal capacity, partly owing to the very frequent deviation of the septum, partly to the very unequal development of the sinuses on either side. I have once observed bony laminae partially subdividing one sinus.

I have seen a sphenoidal sinus, in a female aged 20, measuring $1\frac{1}{4}$ inches in length, the size of a pigeon's egg, having a capacity of 7·6 c.c. This was a large central left sinus, the right sinus being very rudimentary. In one male the capacity of the two sinuses amounted to 10 c.c.

The anatomical relations of the sinuses are of great clinical interest, and explain many of the symptoms that occur when the sinuses are the seat of purulent accumulations. Thus the roof from before backwards is in relation with the olfactory nerves lying in slight depressions, and the frontal lobes, the optic nerves, ophthalmic artery, and the optic chiasma lying in the optic groove, and still more posteriorly with the sella turcica. On

PLATE III.

SKULL (natural size) WITH THE MAXILLARY ANTRUM AND ETHMOIDAL CELLS DISPLAYED.



Skull (natural size) showing the ethmoidal cells (2) (3) (4) (5) opened from the inner wall of the orbital cavity. 1. The lachrymal duct. Below the orbit the maxillary antrum has been laid open. Towards its floor transverse septa (6) (7) are seen traversing the cavity and forming pockets.

N.B. This preparation is displayed in stereoscopic relief in *Plate XVI* at the end of the book.

either side the walls show an eminence corresponding to the carotid or cavernous groove in which lie the internal carotid artery and the cavernous sinus. The anterior surface is formed by the thin sphenoidal turbinated bones which close the sinus on either side, leaving an oval aperture, the *ostium sphenoidale*, situated in the upper and outer angle one or two millimetres below the roof of the nasal fossa. Sometimes the sphenoidal sinuses open into the posterior ethmoidal cells instead. Zuckerkandl has found in a few instances that a horizontal septum divides the sinuses into an upper and lower one, the former then opens into the ethmoidal cells, while the lower opens into the nasal cavity.

Traversing the anterior wall, in a groove near its lower border, is the internal branch of the nasal or spheno-palatine artery; it is, therefore, liable to injury in trephining the anterior wall of the sinus. Here too the spheno-palatine ganglion is situated and may be irritated or compressed in sphenoidal sinusitis, thus explaining the reflex infra-orbital neuralgia and other reflected pains which may lead to an erroneous suspicion of antral or other sources of the pain.

THE LARYNX.

The larynx subserves primarily the functions of respiration, and secondarily, those modifications of expiration which give rise to phonation, to cough, etc. Further it is concerned in deglutition, though its functions are mainly respiratory and vocal.

The mucous membrane of the larynx varies in thickness, and contains numerous acinous glands, especially in the ventricular bands, ventricles, and on the posterior surface of the larynx. On the fibrous vocal cords it is very thin and closely adherent.

The *lymphatic vessels* of the interior of the larynx are very richly developed, and form a network of their own without any free anastomosis with the lymphatics of neighbouring structures, an arrangement which has a most important bearing on the clinically observed facts that intrinsic malignant growths of the larynx tend to remain localised for a long time, and that secondary malignant growths arising by metastasis in the larynx are almost unknown. Von Most has recently investigated this important and debated subject of the lymphatic

supply of the larynx afresh, and states that *the lymphatics of the upper lateral part of the epiglottis, of the ventricle and of the false cords*, empty into two or three larger lymphatic vessels in the neighbourhood of the aryteno-epiglottidean folds where they pass out through the thyro-hyoid membrane to glands which are placed on the "common" jugular vein, at the level of the upper border of the thyroid cartilage. The upper stem frequently passes to the end of the great cornu of the hyoid bone, thence running over the hypoglossal loop to glands on the jugular vein, or in the carotid bifurcation, at the level of the posterior belly of the digastric muscle.

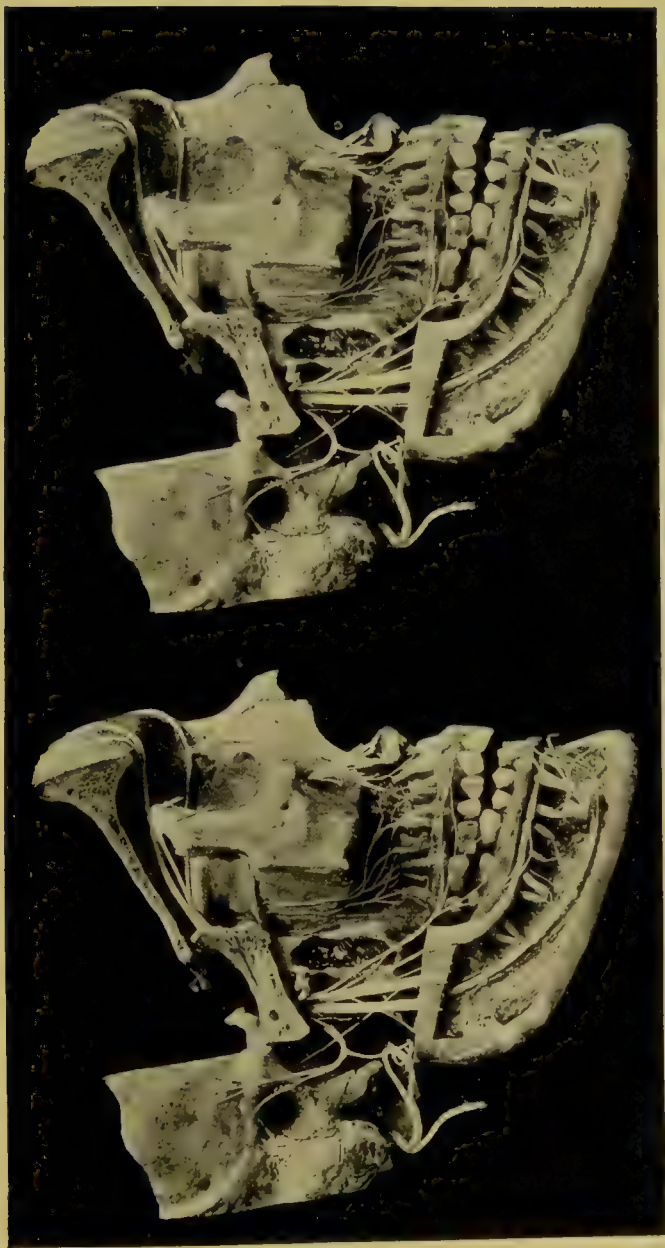


FIG. 9

The lymphatic glands into which the lymphatic vessels of the larynx empty. 1, digastric muscle; 2, sterno-mastoid muscle cut; 3, internal jugular vein; 4, internal carotid artery. (After VON MOST.)

The lymphatic vessels below the vocal cords leave the larynx by piercing (a,) the crico-thyroid membrane, or (b,) the ligamentum crico-trachealis, i.e., either above or below the cricoid cartilage: to

PLATE IV.



Distribution of the fifth nerve. (Stereoscopic and semi-diagrammatic.) Entering the orbital ossa are the frontal, nasal, and lachrymal branches; the supra-orbital branch of the frontal is ascending on the forehead. The posterior dental branches of the superior maxillary nerve and, at its exit from the infra-orbital foramen, the anterior dental, nasal, and labial branches. The pterygoid, the auriculo-temporal, and inferior dental branches of the inferior maxillary nerve are also displayed.

reach glands situated (*a*,) in front of or just below the cricoid, (*b*,) just below the level of the thyroid gland isthmus, or (*c*,) on the jugular vein about the level of the middle of the sternocleido-mastoid muscle.

In some instances these lower lymphatics reach glands lying on the capsule of the thyroid gland and the side of the trachea even as far as the sternum, while branches may even reach the supra-clavicular glands of the neck.

Lymph follicles also are occasionally found in the mucous membrane in various portions of the larynx; thus Dobrowski observed them in the sinus pyriformis in 50 per cent. of larynges, and in some rare cases lymphoid collections resembling solitary follicles in the intestines, while in 8 out of 60 cases there was a regular tonsilla laryngea. Yet in the normal larynx he found that lymph follicles are only occasionally found; they were more frequent in chronic inflammations, and then most frequently on the posterior surface of the epiglottis, and more rarely over the sacculus and in the inter-arytenoid space.

INTRINSIC MUSCLES OF THE LARYNX.

Certain extrinsic muscles act more or less upon the entire larynx; the intrinsic muscles move the different cartilages on one another, modify the apertures of the larynx, and regulate the tension of the vocal cords in respiration, phonation, and deglutition.

All these intrinsic laryngeal muscles are in pairs, except the arytenoideus.

The CRICO-THYROID MUSCLE is a thick, triangular muscle on the front of the larynx, consisting of two parts, the oblique and the horizontal fibres. It *arises* below by a pointed bundle of fibres from the antero-lateral surface of the cricoid cartilage, the fibres diverging, the central *oblique fibres* passing upwards and outwards to be *attached* to the inferior border of the thyroid cartilage. The outer and more *horizontal fibres* pass backwards to be *inserted* into the anterior border of the lower cornu of the thyroid cartilage, some of its fibres being continuous with the inferior constrictor of the pharynx.

An additional slip of fibres, termed the *kerato-cricoid*, is occasionally present, arising from the origin of the lower posticus fibres, and passing upwards and outwards to the posterior margin of the inferior cornu of the thyroid cartilage.

Action.—The oblique fibres raise the anterior arch of the cricoid cartilage towards the thyroid, thus tilting back the arytenoid

cartilages, and rendering tense the vocal cords. (Many anatomists consider the cricoid is the fixed point, and that the thyroid cartilage is drawn down towards the cricoid.)

The horizontal fibres draw the inferior cornu and therefore the whole thyroid cartilage forwards, increasing the tension of the vocal cords.

CRICO-ARYTENOIDEUS POSTICUS MUSCLE, or, more shortly, the *posticus muscle*, is a triangular muscle situated behind the larynx. It arises from the posterior surface of the cricoid cartilage, the fibres converging upwards and outwards to be inserted into the muscular process of the arytenoid cartilage, behind the insertion of the lateralis muscle. The upper fibres are short, and nearly horizontal, the middle run obliquely, and the lower fibres are almost vertical.

Action.—The upper horizontal fibres draw the outer angle of the arytenoid cartilage backwards and inwards, causing the vocal process to rotate outwards, and so abduct the vocal cords and widen the glottic aperture. The vertical outer fibres also draw down the outer angle of the arytenoid cartilage, rotating the cartilage on its joint, and causing the cartilages of Santorini to separate more widely.

CRICO-ARYTENOIDEUS LATERALIS, or, more shortly, the *lateralis muscle*, arises from the upper border of the lateral portion of the cricoid ring, its fibres converging and passing backwards and slightly upwards, to be inserted into the muscular process of the arytenoid cartilage, just in front of the posticus muscle, and to the adjacent part of the outer surface.

Action.—It draws the muscular process of the arytenoid cartilage forwards and downwards, causing the vocal process to rotate inwards, and thus to adduct the vocal cord. This muscle is therefore the physiological antagonist of the posticus muscle, but for the complete approximation of the vocal cords the arytenoid cartilages must be drawn together by the arytenoideus muscle. Inasmuch as the crico-arytenoid joints have an oblique direction outwards and downwards, the inner half of each joint is on a higher level than the external portion, and thus, when the vocal cord is adducted and the arytenoid cartilages apposed in the median-line, it is on a slightly higher level than when it is abducted, and the arytenoid cartilages widely separated.

The THYRO-ARYTENOIDEUS MUSCLE consists of several bundles of fibres which have been named (1,) thyro-arytenoideus externus; (2,) thyro-arytenoideus internus; (3,) ary-vocalis; (4,) compressor sacculus laryngis; (5,) thyro-epiglottideus; (6,) thyro-arytenoideus superior.

It is generally described as consisting of two portions, the superficial fibres being termed the thyro-arytenoideus externus, and the deep fibres the thyro-arytenoideus internus.



FIG. 10.

Diagram to illustrate the action of the intrinsic muscles of the larynx on the vocal cords. Th. C., thyroid cartilage; Cr. C., cricoid cartilage; O., arytenoid cartilages; P., crico-arytenoideus posticus, or shortly, the posticus muscle; L., crico-arytenoideus lateralis; A., inter-arytenoideus; T. A. I., thyro-arytenoideus internus, inserted into the vocal cord at various points, 1, 2, 3.



FIG. 11.

Muscles of the larynx (key to Plate VI). C. A. P., crico-arytenoideus posticus; C. A. L., crico-arytenoideus lateralis; T. A. E., thyro-arytenoideus externus; Ar. Ep., aryteno-epiglottideus muscle.

The EXTERNAL THYRO-ARYTENOID MUSCLE is a broad, fan-shaped layer of fibres *arising* from the lower half of the inner surface of the ala of the thyroid cartilage, close to its retiring angle, and from the crico-thyroid membrane below, radiating backwards to be *inserted* mainly into the outer border of the muscular process of the arytenoid cartilage, by a few fibres into the outer border of the arytenoid cartilage itself, into the thyro-epiglottidean fold of mucous membrane, and into the lateral borders of the epiglottis (a bundle sometimes called the *thyro-epiglottidean muscle*).

Action.—Its upper fibres partially embrace the *sacculus laryngis* (compressor *sacculus laryngis*), hence by their contraction the pouch will be compressed, and its mucoid secretion extruded on to the vocal cords. The lower fibres attached to the arytenoid muscular process cause it to rotate inwards and adduct the vocal cord.

The INTERNAL THYRO-ARYTENOID MUSCLE is a thick bundle of fleshy fibres, triangular on section (well shown in *Plate XXXII*), arising from the retiring angle of the thyroid alæ and the crico-thyroid membrane, passing back in close contact with the vocal cord, along which many of the shorter fibres are inserted, the remainder being attached posteriorly to the vocal process and outer surface of the arytenoid cartilage.

Action.—By drawing forward the arytenoid cartilage the vocal cord is rendered less tense, and by the separate contraction of the short fibres inserted into the vocal cord (postico-ary-vocalis of Ludwig), the elasticity and tensity of different portions of the vocal cord are modified, the part of the vocal cord in front of their attachment being slackened, while the part behind is made tense.

A small third portion (*small thyro-arytenoidæus*) has been described by Sæmmering as sometimes present, as arising from the notch of the thyroid cartilage, and being inserted into the base of the arytenoid cartilage, just above the muscular process, passing internally to the sacculæ in the ventricular band.

Its action is to compress the sacculus internally.

The ARYTENOIDEUS is a single transverse muscle attached to the posterior concave surfaces of the arytenoid cartilage.

Action.—Its fibres draw the arytenoid cartilages together, acting in conjunction with the laterales muscles, and by their swelling push forwards the arytenoid cartilages.

The ARYTENO-EPIGLOTTIDEUS MUSCLE is a thin band of fibres arising from the inferior and outer angle of the arytenoid cartilage, passing obliquely upwards to the upper and outer part of the *opposite* cartilage to which it is partly attached, and thence forwards in the aryteno-epiglottidean fold where it is inserted, and where it joins the external thyro-arytenoid muscle.

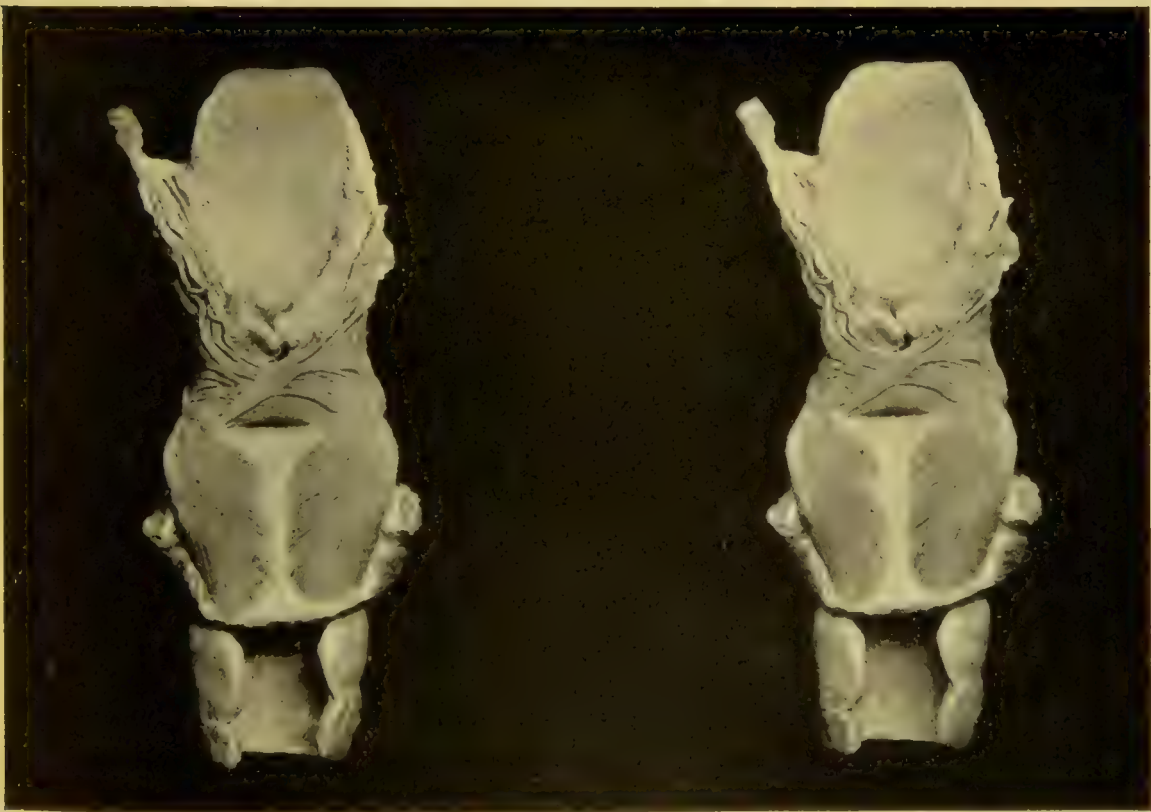
Action.—It draws the arytenoid cartilage and ary-epiglottic fold together.

It is not unusual to describe as a separate muscle the THYRO-

THE MUSCLES OF THE LARYNX.

*Fig. 1.*

The inter-arytenoid muscle has been divided and turned outwards; the right posticus muscle, the attachments of the stylopharyngeus, and the inferior constrictor of the pharynx are also shown.

*Fig. 2.*

The muscles of the posterior surface of the larynx; the crico-arytenoid posticus, the inter-arytenoid, and the aryteno-epiglottidean muscles are displayed.

PLATE VI.
THE MUSCLES OF THE LARYNX.



Fig. 1.

For the names of the muscles shown in this preparation see *Fig. 11*, page 19.

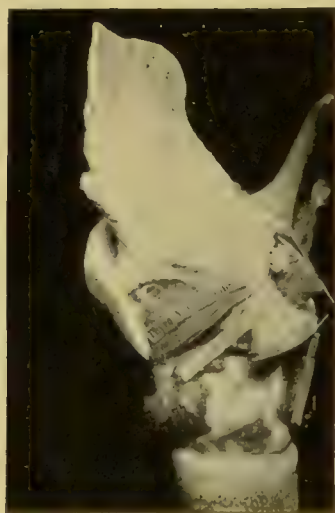


Fig. 2.

The thyro-arytenoideus internus and the crico-arytenoideus lateralis have been laid bare on the left side by removal of the left ala of the thyroid cartilage.

ARY-EPIGLOTTIDEUS, those fibres derived from the oblique portion of the arytenoideus, and those from the thyro-arytenoideus externus muscles which meet in the upper part of the ary-epiglottic folds to be inserted in the epiglottis.

It may be noted that the muscles which lie within the thyroid alæ, *viz.*, the laterales, thyro-arytenoid, transverse arytenoid, and the thyro-ary-epiglottidean muscles form a kind of sphincter, "such as is found in its simplest form embracing the entrance to the larynx of reptiles" (Henle), and these come into play during the acts of coughing, phonation, deglutition.

The *epiglottis* was formerly thought to fold back over the larynx like a lid in the act of deglutition, but Anderson Stuart has shown that the superior aperture of the larynx is closed during swallowing by the close application and forward folding of the arytenoid cartilages in such a way that their apices become closely applied to the cushion of the epiglottis. The older views on the action of the epiglottis during deglutition must be abandoned, for the shape of the epiglottis in many normal individuals is such that it is scarcely conceivable that it should ever be folded back over the larynx like a lid; moreover the epiglottis may be entirely destroyed without swallowing being interfered with. I have examined the larynx of a patient who swallowed some hydrochloric acid, and the only portion of the epiglottis injured was the *laryngeal* surface of the petiolus. Taste buds, too, are found in the laryngeal surface.

THE VOICE.

In the human species the voice is produced by the vocal cords which, as Oertel has demonstrated by means of the laryngostroboscope, vibrate in two planes, *viz.*, vertically and transversely. They thus act both as vibrating reeds and also, so to speak, by producing a string of air bubbles like the syren. Modulation in pitch is brought about by varying the length and degree of tension, and the thickness of the vibrating cords, the glottic chink never being absolutely closed during phonation.

Probably the pitch is also modified in part by the elevation or depression of the larynx which thus varies the length of the vibrating column of air above the glottis.

In birds there are no vocal cords in the larynx, the song-note being produced by the action of the syrinx composed of vibrating reeds which can be approximated and which are situated low down in the trachea close

to the bifurcation, modulation in pitch being brought about by lengthening or shortening the trachea, and thus lengthening or shortening the vibrating column of air.

For the vocal note to be clear the approximation of the cords should be abrupt with the commencement of the sound emitted, and they should at once diverge with the conclusion of phonation. By the more gradual approximation of the cords aspiration is produced, while a similar result follows from the too lagging recession of the cords as the note is finished. Cough is produced by the complete approximation of the cords during a forced expiratory effort, followed by abrupt opening of the glottis. Thus a low note may be possible in conditions which permit of the glottic chink being narrowed, when absolute closure of the glottis during the expiratory effort, and therefore an effectual cough, may be impossible.

The purity and quality of the voice depends not only on the action of the vocal cords, but also on the proper action of the soft palate and the normal patency of the nasal and post-nasal passages.

From a large series of photographs of the laryngoscopic image taken while various notes were being sung, French concludes that, in females at any rate, the larynx may act in a variety of ways in the production of the same tone or registers in different individuals. He found, speaking generally, that in the lower register the whole length of the vocal cord is open, *i.e.*, both the ligamentous and cartilaginous portions of the glottis, the vocal cords being comparatively short and wide in the production of the lower tones, and becoming longer, narrower, and more tense as the voice ascends, at the same time the epiglottis rises, the capitula Santorini are more and more tilted forwards, and the aperture between the posterior portions of the vocal cords increases in size. But at a note in the neighbourhood of E, treble clef, first line, the limit of vocal cord tension is reached (lower break), and the higher notes are produced by the closure of the cartilaginous portions and thus shortening the vibrating cord. At the same time the glottic chink becomes narrower, the epiglottis depressed and the cartilages of Santorini tilted back again. Once more as the voice ascends the vocal cords (ligamentous portion) from being comparatively short and wide, become longer and narrower and more tense, the epiglottis rising, the capitula Santorini being more and more tilted forward and the posterior portion of the glottic chink widening, until, in the neighbourhood of E, treble clef, fourth space, the upper break is reached, when another change occurs. Not only is the cartilaginous glottis always closed, but the ligamentous portion is invariably shortened, the glottic chink being reduced to a very narrow slit, the ligamentous vocal cords often closing in front or behind or both.

INNERVATION OF THE NOSE, PHARYNX AND LARYNX.

INNERVATION OF THE NOSE.

The nervous supply to the nasal passages is derived from the olfactory nerve; *viz.*, filaments of the nasal branch of the ophthalmic to the upper and anterior part of the outer wall and of the septum; filaments from the anterior dental branch of the superior maxillary to the inferior turbinal and meatus; the upper anterior nasal branches of the sphenopalatine (Meckel's) ganglion, and the Vidian nerve, supply the superior and middle turbinals, the intervening meatus, and the upper and back part of the septum; the large anterior palatine nerve supplies the middle and lower turbinals and meatuses, and the naso-palatine supplies the middle of the septum.



FIG. 12.

Diagram of the nerve-supply to the nasal passages. 1, Olfactory bulb with branches descending to the region of the superior and middle turbinated body; 2, sphenopalatine ganglion; 3, Vidian nerve; 4, external filament of the ethmoidal branch of the nasal nerve; 5 and 6, branches of the sphenopalatine ganglion to the superior and middle turbinated bodies; 7, branch of the anterior palatine to the inferior turbinate body; above it is seen the naso-palatine branch to the septum nasi; 8, anterior palatine nerve; 9, middle; and 10, posterior division of palatine nerve.

All these nerves, as well as the sphenopalatine ganglion and the Vidian nerve, are shown in *Fig. 12*, and in *Plate XXXV*. We shall refer to them again in connection with nasal neuroses.

Various functions are subserved by the nerve supply to the nasal passage, *viz.* :—

(a.) Olfaction by the filaments to the olfactory bulb, which

pass through the cribriform plate from the olfactory cells of Max Schultze, from which the fine terminal filaments pass through the external limiting membrane of v. Brunn to lie between the columnar epithelial cells. The olfactory nerve fibres represent the axones of the olfactory cells, and are non-medullated. Von

Brunn showed that the olfactory area of the nasal mucous membrane is restricted to a small area covering the superior turbinal, the adjacent part of the septum and nasal roof, the whole area occupying about six square centimètres.

(b.) Ordinary sensation by ethmoidal branches of the fifth nerve from Meckel's ganglion and the superior maxillary, etc.

(c.) The arterial supply to the mucous membrane and to the erectile tissue of the turbinated bodies is controlled by vaso-motor nerves from Meckel's ganglion, and is under the control of the vaso-motor centres in the medulla, where there is probably a connection with the nuclei of the vagus through association fibres, a physiological connection which has an important bearing on the pathology of various neuroses, as for instance the cases of asthma associated with nasal disease.

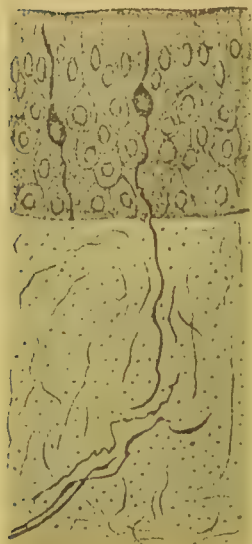


FIG. 13.

Section of the nasal mucous membrane showing the olfactory cells of Max Schultze. After ZUCKER-KANDL.

I would especially direct attention to Cajal's observation that a few of the collateral fibres from the gelatinous substance of Rolando, which is the receptive nucleus of the trigeminus in the medulla, break up under the motor nuclei of the facial and vagus, "and the inference is they communicate" (Golgi method). Also in the mouse Cajal figures collaterals from the sensory ganglia in the substance of Rolando: the descending root of the fifth nerve terminates by arborisation in the substance of Rolando, and from the cells of the substance of Rolando axones arise which terminate in the nucleus ambiguus of the same side and in that of the opposite side.

As a further indication of the physiological association existing between the several portions of the respiratory tract, it is worthy of note that Spencer, corroborating and extending Munk's observations, has demonstrated experimentally the influence of the nasal area, and, in less degree, of excitation of other sensory areas, on normal respiration. He found that four different results were each obtainable from a distinct area on the cortex: all the results being such as can be produced upon

respiration by means of the will : (a,) *Slowing of respiration rate*, or arrest, was obtained from the frontal lobe, just outside the olfactory tract and along the olfactory limb of the anterior commissure ; (b,) *acceleration of respiration rate*, from around the upper end of the infra-orbital sulcus ; (c,) *snuffing*, from the mucous membrane of the nose

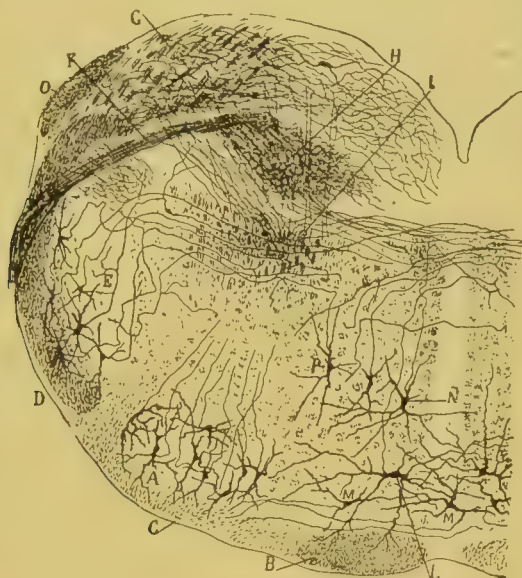


FIG. 14.

Transverse section through the bulb of a new-born mouse (CAJAL). A, nucleus of the facial ; B, pyramids ; D, ascending root of fifth nerve ; E, substantia gelatinosa ; F, sensory root of the vagus and glossopharyngeal ; G, descending root of the vestibular ; H, sensory nucleus of the vagus and glossopharyngeal ; I, lateral central tract of the vestibular, trigeminus, vagus, and glossopharyngeal.

in its upper part, from the olfactory nerves, bulb and tract, and uncinate convolution of the temporo-sphenoidal lobe ; (d,) the fourth effect which can be obtained from the surface of the cerebrum is a widely generalised one, because it can also be obtained from excitation of any sensory nerve. *The chest assumes a position of over-inspiration.*

INNERVATION OF THE PHARYNX.

The fauces and soft palate derive sensation from the second division of the fifth, and from the vagus and glosso-pharyngeal nerves ; the pharynx is supplied by the glosso-pharyngeal.

The motor innervation to the soft palate and uvula, the levator palati and the pharyngeal constrictors are supplied by vago-accessory fibres in the vagus and the pharyngeal plexus. Whether these nerve fibres belong properly to the bulbar portion of the spinal accessory (Horsley and Beevor), or to the vagus, is a debated question (see the arguments for or against the vagus origin of laryngeal motor innervation, *page 30*).

The tensor palati is usually said to be supplied by the fifth nerve.

But in chronic bulbar paralysis, not only is the movement of elevation in abeyance, but the palatal curtain hangs loose and flaccid. It therefore seems probable that its motor nerve centre is in close relation with that of the levator. If the tensor palati muscle is not innervated through the pharyngeal plexus the course of the motor filaments is probably as follows: "They leave the medulla by the accessory nerve and join the vagus, from this they pass to the glosso-pharyngeal by communicating branches, and entering the tympanic branch are conveyed to the small superficial petrosal nerve so as to reach the otic ganglion and from this be distributed to the muscle" (Aldren Turner).

INNERVATION OF THE LARYNX.

The essential functions of the larynx are two-fold, *viz.*, phonation and respiration, the former essentially a volitional or psychological act, governed by the cortical centres, the latter a physiological reflex act, governed by the reflex centres in the medulla. But phonation may sometimes be involuntary and reflex, *e.g.*, in involuntary ejaculations, and respiration may to some extent be controlled or modified by voluntary effort, so that we can only say that the function of phonation is *mainly* represented in the cortex, and that of respiration is *mainly* represented in the medullary centres.

For these two functions there are two separate sets of muscles in the larynx, *viz.*, for phonation, the adductors of the vocal cords which close the glottis, and, for respiration, the abductors of the cords which open the glottis.

The motor fibres for each set of muscles, though running together, are separable into two distinct strands of fibres, both in the recurrent nerve (Russell) and in the internal capsule (Semon and Horsley).

There are separate cortical centres and separate bulbar centres for abduction and adduction of the vocal cords, and each centre is bilateral in action. As the action of each centre is a bilateral one, destruction of one centre produces no corresponding paralysis so long as the other is intact, but irritation or stimulation of either centre will produce bilateral adduction of the vocal cords, *i.e.*, spasm of the glottis. The motor fibres to the larynx are contained in the bulbar accessory roots of the vagus nerve, but whether their ultimate nuclear origin in the medulla is the nucleus of the spinal accessory or vagus, is an

open question, although it seems probable that these bulbar accessory roots belong to the latter.

The recurrent laryngeal nerve is the motor nerve to all the intrinsic muscles of the larynx, except the crico-thyroid which is supplied by the superior laryngeal branch of the vagus and the inter-arytenoideus muscle which receives motor twigs from both the superior and inferior laryngeal nerves. The superior laryngeal nerve is the sensory nerve to the whole of the larynx. This is the distribution of the nerves to the larynx described by Luschka, and is the most generally accepted scheme.

The superior laryngeal nerve contains vaso-motor and secretory fibres to the whole of the laryngeal mucous membrane.

The recurrent laryngeal nerves contain no centripetal fibres, excepting perhaps muscular sense fibres.

Having briefly summarised the subject, in view of the many debated questions in connection with the nerve supply to the larynx, I have thought it well to consider some points in greater detail.

THE CORTICAL MOTOR AREAS.

The experiments of Krause, corroborated by Semon and Horsley and others, have demonstrated that there is in each cerebral hemisphere a bilateral cortical centre for adduction of the vocal cords (as in phonation), and that in the left hemisphere this centre is located in the anterior portion of the lower extremity of the ascending frontal convolution. Thus it forms a part of Broca's speech centre or co-ordinating centre for the labial, lingual and laryngeal muscles, which in man corresponds to the foot of the third frontal and the lower extremity of the ascending frontal convolutions.

Attention has already been directed to the important fact that the cortical laryngeal motor centres are bilateral in action. Thus in motor aphasia due to a unilateral lesion the vocal cords are not affected, though a few observers assert that a unilateral cortical lesion may give rise to unilateral paralysis of a vocal cord. But, as Semon has repeatedly pointed out, no single case corroborated by a post-mortem examination sufficiently complete to demonstrate that the cortical lesion was not complicated by further lesions in either the medulla or in the motor nerve tract has yet been placed on record. I have repeatedly examined laryngoscopically cases of right-sided hemiplegia with motor aphasia and have invariably found the movements of the vocal cords unimpaired, observations which accord with those of Semon, Lermoyez, and several others who have examined the larynx under similar conditions.

Moreover, it was found by Semon and Horsley that the normal

respiratory excursions of the vocal cords in dogs were unimpaired by the entire removal of a cerebral hemisphere, and even of both hemispheres, demonstrating that "the special representation of the respiratory function of the larynx must be a very subordinate thing in the cortex, and that the medullary representation of respiration entirely suffices to keep up the respiratory action of the larynx." Clearly then *no unilateral cortical lesion could result in paralysis of one vocal cord.*

A bilateral cortical centre for abduction was found by Semon and Horsley in the cat, lying close to the border of the olfactory (rhinal) sulcus, yet they failed to localise such a centre in the dog, although the existence of such a centre had been suggested to Semon and Horsley from their discovery of a spot in each internal capsule, excitation of either of which resulted in bilateral abduction of the cords. But Risien Russell, by first dividing the adductor fibres in the recurrent laryngeal nerve and thus excluding the more powerful adductor movements of the vocal cords, has demonstrated the existence of a cortical abductor centre in the dog also, in the anterior composite gyrus just in front of, and a little below, Krause's centre for adduction, and therefore a little in front of and below the anterior extremity of the coronal sulcus. It was sometimes more easily obtained from the prorean gyrus just in front of the point on the anterior composite gyrus, but the two points are quite close to each other and simply separated by the supra-orbital sulcus.

Neither in Russell's, nor in any of Semon's and Horsley's investigations was there obtained the slightest evidence of unilateral representation of the movements of the vocal cords in the cerebral cortex; and in testing this point one recurrent laryngeal nerve was divided transversely when it was found possible to influence the vocal cord whose nerve was intact, with equal ease on stimulation of either cerebral hemisphere.

I may here mention that as the result of his investigations Onodi concludes that the lower limit of the cerebral centre for phonation lies in the corpora quadrigemina, but some experiments of Grabower and, from the clinical standpoint, a case reported by Ransom, seem to disprove Onodi's views. In Ransom's case there were no laryngeal symptoms, although post mortem, the posterior half of the left optic thalamus and the anterior and posterior corpora quadrigemina were involved in the disease (*Lancet*, 1895, I., 1115).

CORONA RADIATA.

Semon and Horsley have demonstrated that, connecting the cortical centres with the medulla, "there exist numerous connecting fibres which can be analysed as they pass down from the cortex through the corona radiata and internal capsule towards the bulb: the arrangement of these fibres in the capsule enumerated from before backwards is: acceleration of respiration, abduction, intensification, adduction." In the monkey acceleration was produced when the fibres immediately in front of the genu and exactly at the genu were stimulated. In the posterior limb at the junction of the middle and posterior fourths of

the capsular border of the middle zone of the lenticular nucleus, they found adduction was very accurately localised, and it was immediately behind this point that Beevor and Horsley located the fibres of the tongue and pharynx.

From the internal capsule the motor fibres descend by a course not yet exactly traced to reach the nuclei of origin of the nerves in the medulla oblongata.

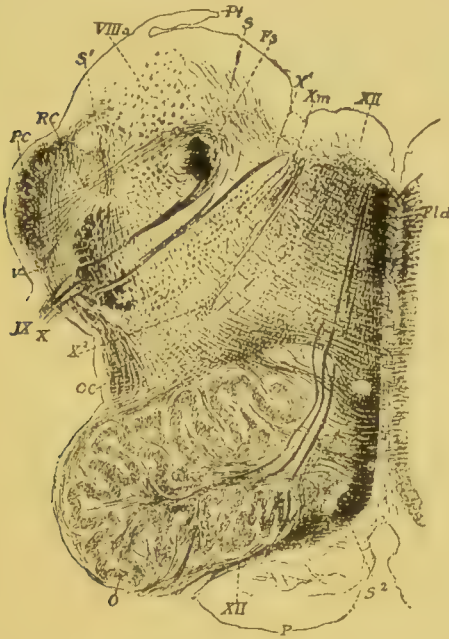


FIG. 15.

Transverse section half through the medulla of a human fœtus at the eighth month. *P*, pyramid; *O*, olive, with the external and internal accessory olives; *IA*, glossopharyngeal nerve; *X*, vagus nerve; *X¹*, nucleus of termination of sensory fibres of vagus; *FS*, fasciculus solitarius; *Xᵐ*, dorsal motor nucleus of vagus; *X²*, nucleus ambiguus; *XII*, hypoglossal nerve and nucleus; *V*, descending root of fifth nerve; *VIIIα*, descending vestibular root. (After KÖLLIKER.)

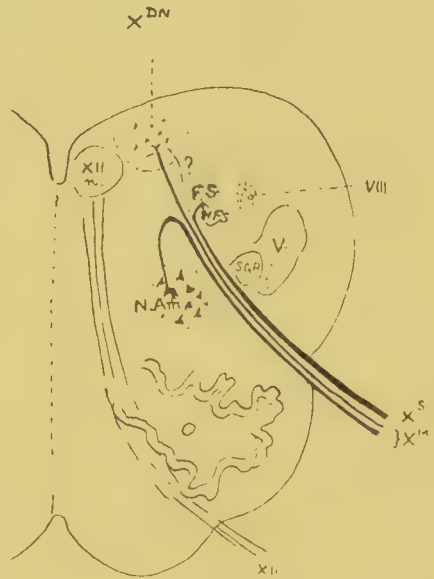


FIG. 16.

Diagram of section through the bulb to show the nuclei of the vagus. *Xᵐ*, motor nerve fibres, some passing out after taking a dorsal turn from the large-celled nucleus ambiguus, *N.Am*, others coming direct from the small-celled dorsal nucleus, *XDN*, just external to the hypoglossal nucleus, *XII n*, in the floor of the fourth ventricle. *X¹*, sensory root of the vagus passing into the fasciculus solitarius, *FS*, to end by arborisation in the nucleus of the fasciculus solitarius, *NFS*. *VIII*, descending vestibular root, *V*, descending fifth; *SGR*, substantia gelatinosa Rolandi; *XII*, hypoglossal nerve root; *?*, indicates the sensory fibres believed by Bruce and Gehuchten to pass to the outer portion of the dorsal nucleus.

THE BULBAR NUCLEI OF THE GLOSSO-PHARYNGEAL AND VAGUS NERVES

The glosso-pharyngeal and vagus nerves having both motor and sensory functions are mixed nerves.

The origin of the *motor* fibres is from the cells lying within the medulla, and probably consists of two parts, *viz.*, (1) a ventral large-celled nucleus, the nucleus ambiguus, and (2) a dorsal small-celled

nucleus, the so called combined nucleus which lies external to the nucleus of origin of the hypo-glossal nerve.

The latter nucleus has been the subject of much discussion, but the general tendency of recent observers is to regard it as motor (it has been suggested that it is a nucleus for unstriated muscle). The sensory nucleus is the fasciculus solitarius or descending root. The sensory nuclei of the vagus are contained, of course, in its root and trunk ganglia—from which axones pass inwards to the medulla.

In a transverse section of the medulla below the level of the striæ acusticæ we may trace the axones of the nuclei of the root ganglia of the vagus (afferent) entering and traversing the bulb till they reach a point posterior to the nucleus ambiguus where they descend, their end tufts being applied to nuclei in the gelatinous substance occupying this area, the descending nerve fibres constituting the fasciculus solitarius.

These views which are upheld by experimental investigations on the glosso-pharyngeal and on the fifth nerve, have been strikingly corroborated from the clinical standpoint by a case recently reported by Bruce. In Bruce's case a tumour, after destroying the fifth nerve, had completely involved the glosso-pharyngeal nerve within the jugular foramen, but had spared the vagus nerve. In the serial sections of the medulla prepared by Marchi's method, it was found that while the whole of the glosso-pharyngeal fibres which enter the upper part of the fasciculus solitarius were degenerated, not a single degenerated fibre could be traced towards the dorsal nucleus or to the nucleus ambiguus. The degenerated fibres were only observed in the upper part of the fasciculus solitarius, the main portion of the lower two-thirds of the fasciculus being formed by the undegenerated fibres of the vagus. Bruce concludes, therefore, that the fasciculus solitarius is an afferent nerve, and in its middle and lower part belongs mainly to the vagus nerve, while the dorsal nucleus and roots from it are motor, as suggested by Forel and Marinesco, and not sensory. But the sensory, ascending, or inferior root, showed degeneration of its fibres.

From observations on rabbits, Van Gehuchten has quite recently declared his conversion to the view that the dorsal nucleus of the vagus is motor in function. He adduces anatomical evidence, having demonstrated and figured axones from this dorsal nucleus to the vagus root in a cat of fourteen days old by the Golgi method, and also pathological evidence conducted by Nissl's method and by Marchi's method.

On the other hand it is necessary to remember that these views are not universally accepted as yet.

It is still an open question whether the vagus nerve or the spinal accessory is the actual channel of innervation to the larynx and soft palate. Much confusion has arisen from the original meaning of the word "accessory" (adhering to) having been lost sight of. Willis in his original description included in the term vagus, all the roots arising from the bulb which have more recently been divided into two roots, *viz.*, the vagus roots and the bulbar roots of the spinal accessory. Under the term spinal accessory, Willis included only the spinal roots, and the nerve was called by him "accessory," not in the sense of "aiding" the vagus but of "adhering" to the vagus. To avoid confusion of terms, I will employ the term spinal-accessory in reference

only to the spinal root or external branch, while the use of the word "bulbar accessory" will leave no doubt in the mind of the reader that the bulbar roots, or internal branch (of the so-called spinal accessory) is alone referred to.

The spinal accessory (or external branch) is a purely motor root supplying the sterno-mastoid and trapezius, and therefore need not further concern us now.

Semon and Horsley, after section of the bulbar accessory in a dog (internal branch of the spinal accessory), observed the vocal cord of the same side at once recede into the cadaveric position, while the autopsy proved the absolute integrity of the corresponding vagus; an experiment which seems to afford conclusive proof that the bulbar accessory roots are the motor nerves for the larynx, at any rate to all the intrinsic muscles except the crico-thyroid. But Grabower, from his experiments on animals, considers that the vagus and not the bulbar accessory is the motor nerve of the larynx.

Even if we accept the view that the bulbar accessory roots contain all the motor fibres of the recurrent laryngeal nerve, we have still to decide whether the nuclei of origin of these roots, that is, the bulbar accessory nuclei, are to be anatomically associated with the nuclei of the spinal accessory or of the vagus. Without committing oneself to either side, we must admit that at the present time the most generally accepted view is that the bulbar accessory nuclei in the medulla are a continuation downwards and form the lower part of the nucleus ambiguus, and are not connected with the spinal accessory. In other words the bulbar accessory roots constitute, according to this view, the lower roots of the vagus, and thus the so-called internal branch of the spinal accessory is really a part of the vagus. In this sense the vagus is the source of innervation to the larynx. Various reasons for regarding the bulbar accessory roots as a part of the vagus, and arising from the same bulbar nuclei, may be cited, such as the following:—

(1,) The spinal accessory nerve of Willis is quite distinct from the vagus in its origin and course, and in its distribution to the sterno-mastoid and trapezius. By careful dissection the spinal nerve can be separated from its adhesions to the lower roots of the vagus, and in sections under the microscope the division between the two sets of fibres is clear. "In the rabbit, horse, mule, ass, and dog, all the roots from the bulb are intimately united, and the spinal accessory runs distinctly apart" (Walter Spencer).

(2,) C. S. Sherrington, in examining the muscular nerves in an amylous human fœtus (the brain and spinal cord being completely wanting), found that the vagus was a large trunk and gave off exactly its usual branches, including the recurrent laryngeal, cardiac and pulmonary. Now the bulbar accessory, like the rest of the vagus was present, but of the spinal accessory inside the dura mater he could find no evidence.

(3,) The spinal accessory nerve, and each of its roots, consists entirely of large medullated fibres, and no ganglion cells are found upon it. But the bulbar accessory roots have the same histological

structure as the rest of the vagus roots, and consist in the main of small medullated fibres with a few large medullated fibres. "The recurrent laryngeal consists of very few grey fibres and medullated fibres up to 11.2μ in diameter" (Edgeworth).

(4.) Pathological evidence in favour of the view that the nucleus ambiguus supplies the levator palati and thyro-arytenoidei interni, is afforded by cases of bulbar paralysis in which these muscles were paralysed during life, atrophy of the cells of the nucleus ambiguus being observed post mortem.

(5.) Batelli, on irritating the roots of the ninth, tenth, and eleventh pairs of nerves in the cat and rabbit (after division of the bulbar accessory nerves of one side) never obtained contractions of the stomach which, however, were well marked when he irritated the cut ends of the bulbar accessory. All the roots appeared to act, but the action and movements of the stomach proved more energetic in proportion as the stimulus was directed towards the more inferior roots. In the dog, fibres belonging clearly to the pneumogastric had no action in the movements of the stomach, but the fibres of the bulbar accessory when stimulated produced movements the more energetic the more the point stimulated descended. In all these animals the spinal roots of the spinal accessory had no action in motility of the stomach. Thus the nerve fibres to the stomach, which it is conceded derives its motor innervation from the vagus, leave the medulla by the so-called bulbar accessory roots, therefore the bulbar accessory roots must belong to the vagus.

On the other hand there is strong pathological evidence in favour of the view that the bulbar accessory nuclei are continuous with the spinal accessory. For example, in a considerable number of cases of bulbar paralysis, which is known to be associated with degeneration of the vago-accessory and hypoglossal nuclei, some or all of the muscles innervated by the recurrent laryngeal nerve together with the muscles of the soft palate have been paralysed (Hughlings Jackson, Stephen Mackenzie, Oltuszewski, Limbeck, Turner), as well as the sterno-mastoid and trapezius, showing that both the spinal accessory and bulbar accessory were affected together, whilst there was no symptom pointing to an affection of the vagus (as distinguished from the bulbar accessory).

A case of syringomyelia with isolated unilateral abductor paralysis of the cords, and paralysis of the trapezius has been recorded by Wientrand.

In yet other cases, the association of spasm of the sterno-mastoid with laryngeal spasm has a bearing on this question. Thus Kellogg (*Med. Rec.*, 1896, p. 219,) reports that "a man insane from alcoholic excess exhibited clonic unilateral spasm of the right sterno-cleido-mastoid, accompanied by loud inarticulate noise." (But echolalia and coprolalia may co exist with this muscular disorder.)

THE SUPERIOR AND RECURRENT LARYNGEAL NERVES.

We have finally to trace the branches of the vagus supplying innervation to the larynx. The superior laryngeal nerve is the sensory nerve to the mucous membrane of the larynx, and by its external laryngeal

PLATE VII.

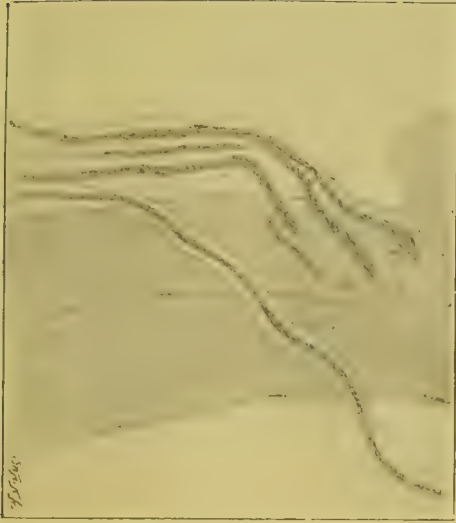


Fig. 1.

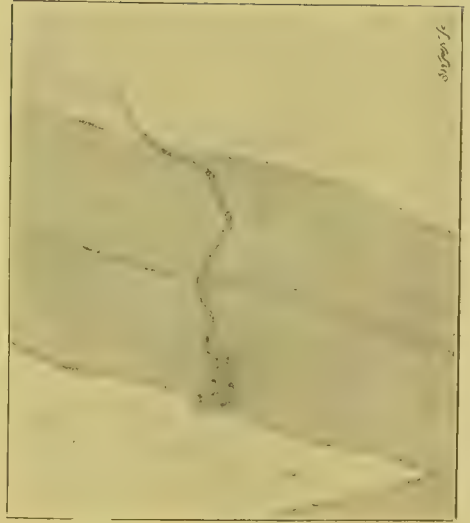


Fig. 2.



Fig. 3.

Fig. 1.—Nerve endings in the abductor muscle (crico-arytenoideus posticus).

Fig. 2.—Nerve endings in the internal thyro-arytenoid muscle.

Fig. 3.—Nerve endings in the lateral crico-arytenoid muscles.

(From preparations by Prof. Grabower.)

branch is the motor nerve to the crico-thyroid muscle. The recurrent laryngeal nerve is the motor nerve to all the other intrinsic muscles of the larynx. The communicating branch between the superior and recurrent laryngeal nerve has been shown by Howell and Huber to be a branch of the former nerve and sensory in function, consisting of a few grey fibres and medullated ones up to 11.2μ . But the recurrent laryngeal nerve is not as formerly supposed, a purely motor nerve, for in the foetus examined by Sherrington and to which I have already referred, the recurrent laryngeal consisted only of fibres from the sensory root ganglia of the vagus, there being not a single motor (ventral) spinal nerve root nor any trace of brain or spinal cord present. The conclusion is that the sensory fibres are muscular-sense fibres, and it is noteworthy that certain experiments by Sherrington and Mott seemed to show that afferent impulses are indispensable to the normal performance of motor acts. But the presence of muscular sense fibres in no way invalidates the conclusions of Semon and Horsley as the result of their experiments, further confirmed by Burger and Onodi, that the recurrent nerve contains no ordinary sense fibres, which Krause believed he had demonstrated.

Exner, from experiments on rabbits, affirms that all the muscles of the larynx except the crico-thyroid and the depressors of the epiglottis are supplied by fibres from both the superior and recurrent laryngeal nerves, the crico-thyroid and depressors of the epiglottis receiving motor fibres from the superior laryngeal alone. He also maintains that the thyro-arytenoideus externus on each side receives motor fibres from both recurrent nerves, having traced in microscopical sections nerve fibres from one side to their termination on the opposite side. Inasmuch as the recurrent laryngeal nerves contain muscular sense fibres (see above), it is very possible that Onodi's suggestion that Exner's crossed fibres were of this character and not motor fibres, is correct; Exner further states that there is considerable variation in individuals in the motor innervation of the larynx. But Exner's conclusions do not accord with clinical evidence, and therefore it is impossible at present to regard them as representing the motor innervation of the larynx in man.

Vaso-motor innervation of the larynx.—The existence of vaso-motor nerve fibres in the superior laryngeal nerve of the dog has been investigated experimentally by Spiess and Hédon; the observations of Spiess were negative, but Hédon has demonstrated that the superior laryngeal nerve contains vaso-dilator fibres for the laryngeal mucous membrane, stimulation of the peripheral end of either divided superior laryngeal nerve causing vascular dilatation in the mucous membrane of the corresponding half of the larynx, including the anterior and posterior surfaces of the epiglottis. The effect is unaltered by previous division of the two inferior laryngeal and vago-sympathetic nerves proving that the effect is produced by stimulation of the superior laryngeal nerves alone. Hédon is inclined to believe that the superior laryngeal nerve also contains tonic vaso-constrictor fibres (by the inhibition of which on stimulation of the vaso-dilator fibres, the vascular dilatation is effected). Simple section of the nerve of one side appeared to cause the mucous membrane of that side to become a little more vascular; moreover, after section of the vago-sympathetic at the middle of the neck, he observed a slight hyperemia of the laryngeal mucous membrane of the arytenoid and epiglottis, while on stimulating the central end of this nerve

the redness was apparently lessened. On the other hand, ablation of the superior cervical ganglion, or division of the recurrent nerve and stimulation of its central end gave negative results; thus he inclines to the view that the vaso-motor nerve fibres are confined to the superior laryngeal nerve.

Secretory innervation.—Hédon has shown that after gently drying the mucous membrane of the larynx of the dog or sheep and stimulating the peripheral end of the divided superior laryngeal nerve, the posterior surface of the epiglottis, the arytenoid and the subglottic mucous membrane, of the corresponding side, secreted drops of mucus forming globules at the orifice of the glands, the other side remaining dry.

Kokin has also pointed out the secretory action of the superior laryngeal nerve on the mucous glands of the larynx of the dog. Moreover he found that in the dog the superior laryngeal nerve contained secretory fibres for the superior and inferior portions of the trachea, and that these fibres joined in the larynx with the inferior laryngeal.

This latter observation is interesting in connection with the fact established by Howell and Huber, that the communicating branch between the superior and recurrent nerve is sensory and not a motor branch, for the other vaso-dilator nerves which have been investigated are sensory nerves, *e.g.*, the glosso-pharyngeal for the mucous membrane at the base of the tongue; the lingual for the anterior two-thirds of the tongue; the superior maxillary for the nasal, and the superior and buccal branch of the inferior maxillary for the mucous membrane of the cheeks, lips and gums.

DEVELOPMENT OF THE NOSE, PHARYNX, AND LARYNX.

Some reference to embryology is necessary to explain the congenital defects and other pathological conditions of practical interest that are referred to later. For the following description I am mainly indebted to the work of Prof. His.

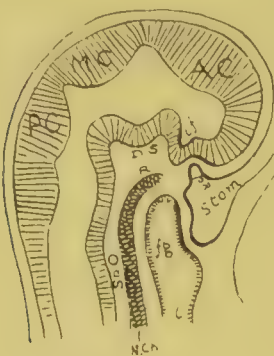


FIG. 17.

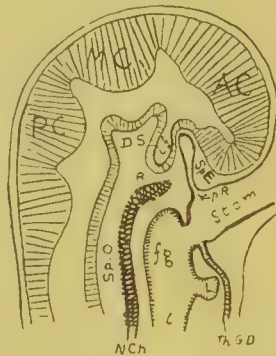


FIG. 18.

Stom., Stomodæum; *fg*, foregut; *P.R.*, pouch of Rathke; *if*, infundibulum; *Th.G.D.*, Thyroglossal duct. Other parts are described in the text.

The diagrammatic sections of the head in an early embryo show the three primary encephalic vesicles: (1.) the anterior cerebral vesicle (thalamencephalon); (2.) the mid-cerebral vesicle

(corpora quadrigemina and peduncles of cerebrum); and, (3,) posterior cerebral vesicle (cerebellum and medulla). Between the anterior and middle cerebral vesicles is the first cephalic flexure (*DS*), corresponding in position to the sella turcica of the body of the sphenoid, marking the division between the pre-sphenoid and post-sphenoid parts, the cavity in the primitive brain here representing the third ventricle. Below we see the anterior extremity of the notochord, embedded in the investing mass of Rathke (*R*). The latter forms the parachordal cartilage containing (*a*,) the post-sphenoid (*Sp.O.*), and (*b*,) pre-sphenoid (*Sp.E.*) developmental centres from which respectively, develop (*a*,) the bones of the cranial base as far forward as the sella turcica—that is to say the spheno-occipital portion of the basis cranii, and (*b*,) the pre-sphenoid or spheno-ethmoid portion.

Primitive mouth.—By an invagination of the epiblast the primitive mouth or stomodæum is formed which dips in until it meets the anterior end of the foregut (the future pharynx). By absorption of this partition, the cavity of the mouth becomes continuous with that of the pharynx.

The pouch of Rathke.—A diverticulum from the stomodæum of the embryo commences to form prior to the communication between the stomodæum and foregut, and soon passes up between the pre- and post-sphenoid developmental centres to the sella turcica (*F.g. 17; P.R.*). There it meets a similar pouch, the infundibulum (*if.*) from the third ventricle of the brain, which comes to form the posterior lobe of the pituitary body, the anterior lobe being formed from the pharyngeal pouch just described.

The lower portion of the pouch of Rathke closes later in foetal life, but its position is marked in after life by the median furrow of the pharyngeal tonsil, the *recessus medius*, and sometimes a short tubular recess is left, the bursa pharyngea, or pharyngeal pouch. Thus the pouch or median recess is merely a pit or furrow in the rhino-pharyngeal tonsil, and has no clinical significance apart from that tonsil (see *Plate VIII*).

The pharyngeal tonsil is formed by invagination of the hypoblastic epithelium in longitudinal folds around the orifice of the pharyngeal pouch, lymph follicles being developed from the mesoblastic tissues between the epithelial invaginations. The pouch of Rathke develops in the front part of the stomodæum

and at birth its vestigial orifice, surrounded by the pharyngeal tonsil, has shifted its relations, coming to lie beneath the body of the spheroid. After birth the tonsil continues to take a more posterior position. Killian observes that "the pharynx tonsil of man moves between the sixth month of embryonic life and end of the second decennium, from the baso-sphenoidal to the basi-occipital region."

Thyro-glossal duct.—Here we may conveniently refer to the origin of the thyro-glossal duct. Just as the pouch of Rathke arises by a diverticulum from the stomodæum, so the thyro-glossal duct, according to His, forms by the evagination of the

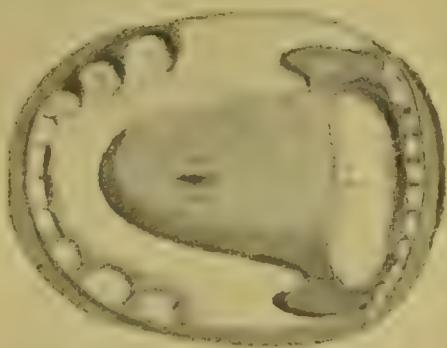


FIG. 19.

To illustrate the development of the larynx and the formation of the thyro-glossal duct. T, anterior segment of the tongue; E, portion of the posterior segment from which the epiglottis is developed; PLA, plica arytenoidea, forming the posterior wall of the larynx and arytenoid cartilages; the position of the foramen cæcum at the upper extremity of the tractus thyroglossus is indicated, F.C. and more posteriorly the evagination which forms the supra-glottic portion of the laryngeal cavity. The infraglottic portion of the larynx is formed by the upward extension of a diverticulum, L, from the foregut (see Fig. 18); the imperfect absorption of the intermediary septum leads to congenital web formation shown in Fig. 32. (His.)

hypoplast at the back of the anterior segment of the future tongue, which descends. From the lower portion the rudimentary median lobe of the thyroid gland is developed, the upper part of the duct being the *ductus lingualis*, the lower part the *ductus thyroidei*. The duct obliterates, leaving as vestiges the foramen cæcum at the back of the tongue, and below, the cornu medium of the hyoid, and various bursæ. Pathological evidence of this thyro-glossal duct is seen in the appearance of an accessory thyroid at the back of the tongue, and in cysts which sometimes appear in the median line over the thyroid cartilage.

The lingual tonsil is formed by the invagination of the hypo-



blastie epithelium in the neighbourhood of the thyro-glossal diverticulum, and by the lymph follicles being developed around the primitive crypts formed by the epithelial invaginations.

We may now turn to another phase in embryonic development, when two sets of plates or arches have appeared above and below the stomodæum or oral fissure, those in front of the oral fissure being termed the *pre-oral arches*, those behind the oral fissure the *post-oral arches*.

THE PRE-ORAL ARCHES.

The Face and Nose, above the mouth, are developed from the pre-oral plates, viz., on each side a median fronto-nasal, and a lateral maxillary plate. The median fronto-nasal plates extend downwards as far as the oral fissure, their lower ends dividing so as to surround the primitive nasal pits.

In negroes the nasal orifices remain widely separated, and the imperfect union of these plates in the middle line may cause in after life a depression, or a bifid tip to the nose (see *Fig. 47*, p. 57).

Development of the internal nose.—We have already referred to the two developmental centres in the parachordal cartilages, the pre-sphenoid and the post-sphenoid. Cartilaginous trabeculæ from the pre-sphenoid centre on either side extend forwards, meeting in the median line to form the cartilaginous septum nasi and at their anterior extremity surrounding the nasal pits.

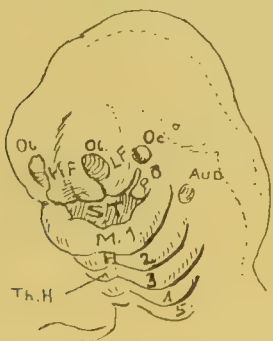


FIG. 20.

Diagram of the head of a fetus, illustrating the formation of the external nose, the mouth, and the pre-oral and post-oral plates.

Ol, Olfactory pits; *MF*, median-frontal and *LF*, lateral frontal plates; *ST*, Stomodæum; *M*, mandibular arch; *H*, hyoid arch; *Th.H*, thyro-hyoid arch, etc.

(From models in Wax by Ziegler in the Museum of the Bristol University College.)



FIG. 21.

Head of more developed foetus, the nose still presenting the central depression and negro-type, while the remains of the hyoid fissure still persist.

The imperfect union of these cartilages may result in a split septum in after life.

The nasal pits appear to form the vestibule, and are at first closed at the bottom by the layer of involuted epithelium. This epithelial septum disappears so as to form a free passage from the nasal orifices with the nasal fossæ.

If this septum is not absorbed completely, a partial or complete septum remains in after life at the back of the vestibule.

POST-ORAL ARCHES.

The appearance of horizontal fissures in the lateral walls of the foregut results in the formation of a series of five bars or arches, with intervening clefts.

The visceral plates or clefts. From the first arch or plate, the lower jaw is developed; hence it is called the mandibular arch.

From the second arch or plate, the anterior pillar of the fauces and a portion of the hyoid bone is developed; hence it is called the hyoid arch.

The cleft between the first and second arches, *i.e.*, the first visceral or hyo-mandibular cleft enters into the formation of the Eustachian tube and middle ear, and more posteriorly the external meatus.

The remaining three persist in fishes, etc., forming the gills, hence termed by Rathke the branchial arches and clefts. Only the first of these, *i.e.*, the third arch, concerns us here. From the third arch the posterior pillar of the fauces is developed, and the cleft between the second and third arches forms the fossa of Rosenmüller above, and the sinus tonsillaris below.

Formation of the palate, pillars of the fauces, and tonsil.—The palate is formed by the ingrowth of palatal plates from the upper jaw, which meet in the median line. According to His, these plates extend backwards, crossing the second and third visceral arches and the intervening cleft, so as to divide the foetal pharynx into an upper and lower series of parts.

In the upper series—*i.e.*, *above the palate*—we have the Eustachian tube developed from the cleft between the first and second visceral arches. Behind it is the fossa of Rosenmüller corresponding to the cleft between the second and third visceral arches, and in the same cleft *below the soft palate* (the sinus

tonsillaris) the tonsil develops, the unoccupied portion of the cleft above the tonsil being the supratonsillar fossa.

The faucial tonsil arises by an invagination of the hypoblast in the sinus tonsillaris. The diverticulum thus formed subdivides, and lymphoid tissue is formed around the primitive crypts. In early foetal life the anterior palatal pillar widens and extends backwards, forming a triangular fold partially covering the cleft in which the tonsil develops. In this way the anterior surface of the tonsil and the supratonsillar fossa may become more or less completely covered by the thin free border of the plica triangularis. Paterson's researches have shown that the supratonsillar fossa may extend downwards so as to admit a bent probe between the outer side of the tonsil and the superior constrictor of the pharynx, even as far as the inner surface of the lower jaw, while it may likewise extend upwards and backwards into the soft palate. The immense clinical importance of this supratonsillar fossa and the special character it imparts to the faucial tonsillitis will be considered later; it is sufficient here to direct attention to: (1,) the existence of this fossa; (2,) to the fact that some of the uppermost crypts of the tonsil open into it; (3,) that its orifice of communication with the cavity of the mouth, while generally large and free, varies greatly; and, (4,) that from its extent in different directions the fossa may form a trap for micro-organisms and the accumulation of decomposing caseous masses extruded from the upper tonsillar crypts.



FIG. 22.
The supra-tonsillar fossa.

CHAPTER II.

*EXAMINATION OF THE PHARYNX, LARYNX,
NOSE AND EAR.**GENERAL SEMEIOLOGY.*

FOR the examination of the pharynx and larynx one requires: (1.) a concave reflecting forehead mirror; (2.) a laryngeal mirror—about the size of a half-penny, and mounted on a handle; and (3.,) for satisfactory examination a brilliant light is essential. Bright daylight answers admirably when it is available, the patient being placed with his back to the light. But it is generally more convenient to employ artificial light, which is under better control. Any good lamp will do, or even a candle, provided the room be darkened, but special lamps give a much better illumination of the parts to be examined, and for posterior rhinoscopy are almost a necessity. Without attempting to describe in detail the many forms of lamps and brackets, I may mention Morell Mackenzie's gas bracket, with an argand burner and a bull's-eye condenser to collect the rays, as one of the most cleanly and convenient. If fitted with an eighty-candle power Welsbach incandescent burner, with the adjustment for rotating

it on its axis, it should cost under £4, and we then have a light which, for brilliancy and whiteness, is only excelled by the oxyhydrogen lime-light. If cost is no great obstacle, the oxyhydrogen lime-light offers some advantages over the Welsbach.

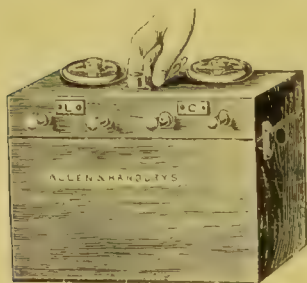


FIG. 23.

A convenient Portable Accumulator for electric light and cautery.

It is very essential that the light should be freely moveable in every direction, so as to allow of ready adjustment in focussing the light on the part to be examined. If a small electric lamp be used on the forehead mirror, all other lamps may be dispensed with for use

at patients' houses. A small portable accumulator, weighing only a few pounds, will keep such a lamp illuminated for several hours in the aggregate. But when exhausted it can only be re-charged by an electrician having a suitable dynamo, and in the country some trouble and delay would be involved in getting the re-charging done, though the actual cost of re-charging is insignificant.

The reflecting mirror should be of about 14-inch focus, and it should have an opening in the centre, through which the eye of the observer looks. It is fixed to the forehead by means of a band round the head or a metal band over the head, such as Fox's head-band, or carried on a spectacle frame. It is essential that the central opening should come immediately in front of the observer's corresponding pupil, and that the mirror should be freely adjustable.

PHARYNGOSCOPY.

In examining the pharynx the examiner sits facing the patient with the reflecting mirror adjusted either in the front of the centre of the forehead, or over the right eye in such a position that the eye looks through the aperture in the centre. The lamp is placed on either side, usually the patient's left, about four inches from the patient's head, on a level with his ear, and so that the light is directed to the reflecting mirror on the examiner's head, and thence into the patient's mouth. It is generally necessary to depress the tongue either with a tongue depressor, spoon, or spatula. Fränkel's is a very handy tongue depressor, the under surface of the extremity is serrated so as to grasp the tongue, while the fenestra may be utilised to draw forward the uvula in inspection of the posterior pharyngeal wall. Türck's will be found useful in some cases, as the handle is twisted to the left of the median line so that the left hand does not come in the way of the right when using the rhinoscope. In using the tongue depressor it should be placed well over the arch or dorsum of the tongue, and at first only gentle pressure should be used. If we attempt to depress the organ suddenly and forcibly, it will be involuntarily arched up.

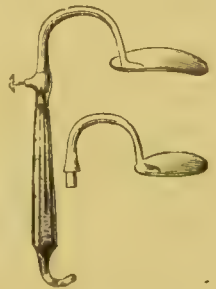


FIG. 24.
Türck's Tongue
Depressor.

For clinical purposes the pharynx is divided into three regions, the rhino-pharynx (or naso-pharynx), the oro-pharynx, and the laryngo-pharynx.

The *rhino-pharynx* is continuous with the anterior nasal cavities, and extends from the base of the occiput and sphenoid downwards as far as the isthmus, the narrow space corresponding to a line drawn from the posterior margin of the soft palate to the posterior pharyngeal wall. Into it open the Eustachian tubes by their trumpet-shaped orifices, from the posterior margin of which may be seen the salpingo-pharyngeal folds extending downwards and leaving on each side a fossa between them and the posterior wall of the pharynx, the fossa of Rosenmüller. The orifices of the Eustachian tubes are just behind the posterior extremity of the inferior turbinated body.

The mucous membrane is covered with ciliated columnar epithelium, and is more abundantly supplied with mucous glands than the anterior nasal cavities. Numerous lymph follicles exist throughout the pharynx, and a collection of these in the roof and posterior wall of the rhino-pharynx forms a mass, similar to the faucial tonsils, termed Luschka's or the pharyngeal tonsil. The pharyngeal tonsil presents an uneven surface with longitudinal ridges. Towards the lower portion the elevated *bursa pharyngea*, with a central depression, the "foramen," may be observed in some individuals.

The *oro-pharynx* lies between the isthmus above, and a horizontal line on a level with the top of the epiglottis.

The *laryngo-pharynx* extends down to a point corresponding with the cricoid cartilage where the œsophagus commences, and includes the hyoid fossæ and parts generally described with the larynx.

The tonsils lie between the anterior and posterior pillars of the fauces, *i.e.*, in the *plica triangularis*.

First, the condition of the parts during quiet breathing should be noted. The tonsils should not normally project beyond the free margin of the faucial pillars, though it is by no means uncommon to find some enlargement in persons who do not complain of any throat trouble whatever. Similarly a considerable variation from the usual pink, smooth character of the mucous membrane of the pharynx must be regarded as within the limits of the normal.

The patient should then be directed to sound *ah!* and the presence or absence of the normal retraction of the soft palate noted. While observing the colour and contour of the *velum palati*, we must guard against overlooking any diseased condition on its posterior surface, for, especially in syphilis, extensive ulceration may affect its posterior surface only, and nothing strikingly abnormal appear on the anterior; yet, on careful observation, we usually find sufficient diffuse hyperæmia with deficient mobility of the soft palate to put us on our guard in such conditions. The soft palate and uvula are rich in small mucous glands, and sometimes these appear as small elevations the size of millet seeds. It is desirable to make the patient "gag" or retch before the examination is concluded, as by this means the pharyngeal muscles bring into view the lateral walls behind the tonsils, disclosing any thickening such as is found in *pharyngitis lateralis*, and sometimes showing a considerable hypertrophy of the tonsils previously unobserved.

Among congenital deformities that may be encountered we may mention perforation of the anterior faucial pillar (*Fig. 25*), bifid uvula, absent uvula or soft palate, divided soft palate (cleft palate) and a pharyngeal pouch.



FIG. 25.

Congenital perforation of the anterior faucial pillars.

The examination of the rhino-pharynx is described under the examination of the nose, and need not be referred to now.

LARYNGOSCOPY.

The *face* of the laryngeal mirror must be warmed over the lamp, so as to prevent the breath from condensing on it and obscuring the image. If an electric lamp or sunlight is used, and the mirror cannot conveniently be warmed, a very thin film of soap on the surface of the mirror prevents its being clouded over.

The patient should sit up with the body slightly inclined forwards and the head slightly tilted back. Then the examiner, having tested the temperature of the mirror on the back of his hand, directs the patient to open the mouth widely, breathe quietly, and to protrude the tongue, which is gently but firmly

grasped in a small towel by the physician's left hand. The laryngeal mirror should be lightly held in the examiner's right hand as one holds a pen, steadily but gently pressing against the uvula and soft palate, yet not so far back as to actually touch the posterior wall of the pharynx.

At first, perhaps, only the dorsum of the tongue and the lingual surface of the epiglottis may appear reflected in the laryngeal mirror, but by altering its angle, or placing the mirror farther back, the other parts will be successively brought into view. While keeping the mouth widely open, the patient should be directed to sound *eh! eh!* (not *ah!*), thus causing the larynx to be raised and brought more readily into view. The vocal cords can then be seen approaching and diverging alternately in phonation and inspiration.

It will be noticed that the laryngeal image is inverted, since it is seen reflected in the laryngeal mirror. The reason for this inversion will be obvious from the accompanying diagram, which

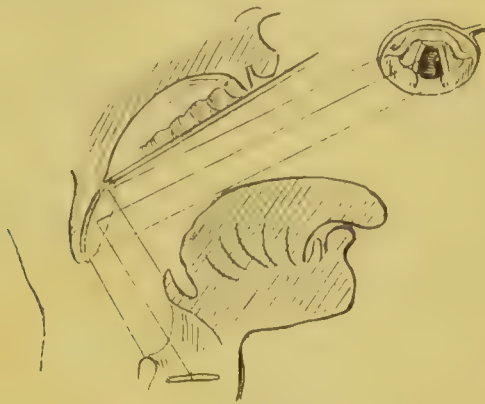


FIG. 26.

Diagram to show the position of the Laryngoscopic Mirror which will give the most perfect view of the larynx, and to explain the inversion of the laryngeal image.

shows that the anterior or upper portion of the mirror is lying immediately over the epiglottis, while the posterior or lower part of the mirror is above the posterior half of the larynx. Thus it is that the anterior portion of the larynx (epiglottis, etc.) is seen reflected in the upper half of the mirror. The relative position of the parts on the right and left remains, of course, unaltered.

Clinically, we distinguish three portions of the larynx, (1,) the supra-glottic, or the part above the ventricular bands; (2,) the

The Normal Larynx, Nose, and Rhinopharynx.



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 5.



FIG. 6.

- FIG. 1.—Laryngoscopic image of a normal female larynx during quiet respiration. The vocal cords are lying midway between adduction and abduction,—the position of quiet respiration.
- FIG. 2.—The same during vocalisation. The vocal cords are adducted and the arytenoid cartilages are brought into apposition.
- FIG. 3.—A normal male larynx on deep inspiration, showing the vocal cords widely abducted, and the bifurcation of the trachea.
- FIG. 4.—Explanatory diagram of the laryngoscopic image.
- FIG. 5.—View of the nasal passages by anterior rhinoscopy.
- FIG. 6.—The posterior rhinoscopic image in a healthy adult.

glottic part, between the ventricular bands and the vocal cords, including the ventricles, and (3,) the infra-glottic, extending from the vocal cords to the lower border of the cricoid cartilage.

The Laryngoscopic Image (see *Plates LX and X*).—The most striking object is the epiglottis, which, from its being bent on itself, shows portions of both the upper and lower surfaces. The epiglottis normally varies greatly in shape and position in different patients. In some it is erect and only slightly curved; in others it overhangs the larynx, or is very much curved and curled.

Behind the epiglottis are the diverging pearl-white vocal cords passing backwards to be attached to the arytenoid cartilages. The cartilages of Wrisberg and Santorini are seen as rounded swellings in the lower part of the image, and forming in part the posterior boundary of the larynx, and between the arytenoid cartilages is the inter-arytenoid space. The ary-epiglottic folds of mucous membrane pass from the arytenoid cartilages, on either side, forward to the epiglottis.

The true vocal cords, essentially the ligamentous portion of the thyro-arytenoid muscles, are attached posteriorly to the *processus vocales* and to the anterior surface of the arytenoid cartilages, and, passing forwards, are attached in front in the angle of the thyroid cartilage, forming the anterior commissure, just below the projection or thickening termed the cushion of the epiglottis.

Between the true vocal cords is the opening of the larynx, the *rima glottidis*, and external to the vocal cords, and lying on a higher level are the pink false cords or ventricular bands. In most cases, especially by tilting the mirror laterally, the opening of the *sacculus laryngis*, or ventricle of Morgagni, can be seen on either side between the ventricular band and the cord. A few rings of the tracheal cartilages can generally be seen below the vocal cords, and rarely, by directing the light well down through the rima, the division between the right and left bronchi may be made out.

The epiglottis is attached to the base of the tongue by three ligamentous folds, the one central (superior glosso-epiglottic lig.), and the right and left lateral glosso-epiglottic folds. The spaces between these folds are named the *valleculæ*.

The opening of the upper end of the œsophagus lies between

the posterior margin of the larynx and the posterior wall of the pharynx ; but though shown as an actual opening in the diagram, the larynx lies in contact with the posterior wall of the pharynx, except during the passage of food. On either side of the larynx are the hyoid fossæ.

In making a laryngeal examination, first observe the larynx during quiet respiration (*Plate IX, Fig. 1*), noting whether the colour of the mucous membrane is healthy, and the form of the various structures normal, and free from swelling or ulceration. The epiglottis is slightly yellowish, and the rest of the laryngeal mucous membrane is pale pink. The alteration in the colour of the laryngeal mucous membrane may be rapid ; thus a pale and anæmic mucous membrane, on a first inspection, may give place to a normal tint, or even hyperæmia, from vaso-motor changes during subsequent introduction of the mirror. The vocal cords should be pearly white, or very slightly pink, and the free margins perfectly smooth and even, though in those who use the voice much, especially in bass voices, the cords may be persistently red without being abnormal. They should lie symmetrically midway between abduction and adduction, (the position of rest or quiet respiration), at one time called the cadaveric position, because it was thought to be the position assumed by the vocal cords in the dead body. Semon has shown, however, that in the position of rest, owing to reflex tonus of the abductors, the glottic chink is wider than in the cadaver or after section of the recurrent laryngeal nerves.

Next we note the movements of the vocal cords during vocalisation (*Plate IX, Fig. 2*) and deep inspiration (*Plate IX, Fig. 3*). On phonating *ch! ch!* the vocal cords should approach till the free margins almost meet in the middle line, the arytenoid cartilages at the same time being also approximated by the arytenoid muscle so as to obliterate the inter-arytenoid space.

In deep inspiration the vocal cords are widely abducted, and diverge considerably more than during quiet respiration, the inter-arytenoid space being correspondingly increased.

Thus we may distinguish four positions of the cords, viz.—

- (1.) Adduction, or phonatory position.
- (2.) Position of quiet respiration.
- (3.) Abduction, or position in deep inspiration.
- (4.) Cadaveric position.

Semon, who has investigated the size of the glottic chink in the varying positions of the cords, states that the average width of the glottis in a healthy adult man during quiet respiration is 13.5 mm., whereas after death the maximum width ever seen so far is 6 mm. in a man and 5 mm. in a woman. The average in men is 5 mm. and in women 4 mm. The minimum cadaveric width (in either sex) is 2 mm.

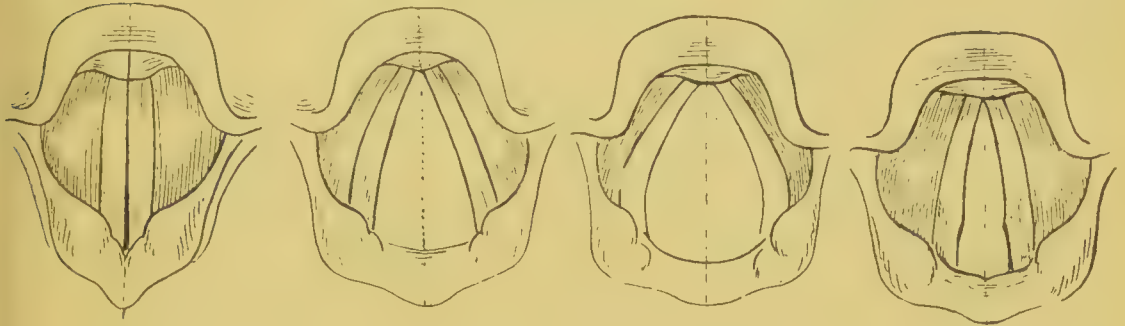


FIG. 27.—Adduction.

FIG. 28.—Quiet Respiration.

FIG. 29.—Abduction.

FIG. 30.—Cadaveric position.

Finally, it may be necessary to test the tactile sensibility of the larynx by means of a curved probe. A normal larynx is very sensitive, and violent cough is set up on touching it; but if anæsthetic, this does not occur on probing.

Difficulties in Laryngoscopy.—We may fail to obtain a good laryngoscopic image, either from faulty manipulation, or owing to the excessive irritability or the peculiar conformation of the larynx:—

(1,) A common fault is to hold the laryngoscopic mirror at the wrong angle, and too far forward, so that only the dorsum of the tongue and the anterior surface of the epiglottis is reflected in it (*Fig. 31*). By placing the mirror somewhat further back and less horizontally, as shown in *Fig. 26*, a more complete image is obtained.

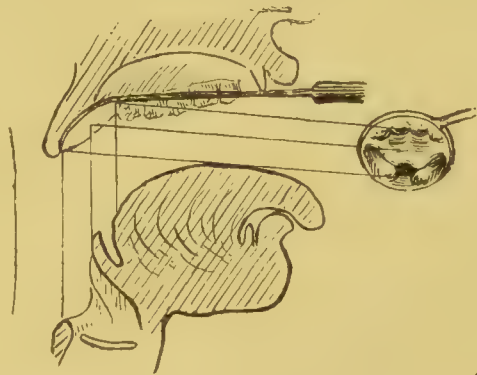


FIG. 31.

Diagram to show the faulty position of the Laryngoscopic mirror, which will not give a view of the vocal cords.

(2,) In introducing the mirror clumsily the fauces may be unnecessarily titillated, gagging and retching being started.

This is especially liable to occur if the posterior pharyngeal wall be touched with the mirror.

(3.) Gaggling and retching may be due to a hyperæsthetic condition of the pharyngeal mucous membrane, in which case painting or spraying the fauces with a weak solution of cocaine (5 per cent.), or sucking ice, may be tried:

(4.) The dorsum of the tongue may rise so much that either the mirror cannot be introduced, or its reflecting surface is out of view. If forcible *protrusion* of the tongue by the patient, or taking a deep inspiration, does not overcome this difficulty, the patient should hold his own tongue while the examiner depresses the dorsum with a spatula in the left hand. Do not drag on the tongue, nor press it unduly on the lower incisors. In all cases let the patient protrude it, and then simply seize and hold it firmly in position.

(5.) The patient may be tongue-tied and protrusion impossible. In this case the frænum should be snipped, or the dorsum may be simply depressed with a spatula.

(6.) In some cases, when laryngoscopy by the usual method of holding the tongue has not been satisfactory, one is able to obtain a good laryngoscopic view by simply depressing the tongue with a spatula as it lies in the mouth.

(7.) If the tonsils are greatly enlarged, and prevent the introduction of the usual mirror, a smaller one should be used.

(8.) As already stated, the natural conformation of the epiglottis varies greatly. It may be so pendulous as to overhang the larynx, so that only its anterior surface is seen, the larynx, or all but its posterior margin, being out of sight. There are several ways of overcoming this difficulty. In slighter cases the act of phonating *ee! ee!* or coughing with the mirror *in situ*, may suffice to raise the epiglottis, when the vocal cords may come into view. Failing by this manœuvre, direct the patient to throw his head well back, place the mirror nearer the posterior wall of the pharynx, and somewhat more vertically than usual, the observer's eye being well above the level of the patient's mouth. In a few cases it is only possible to see the vocal cords by raising the epiglottis with a retractor.

Lastly, the patient may hold the breath from nervousness; a few instructions and a little patience will soon overcome this difficulty. Moreover, it is important to remember that, in ner-

PLATE X.

The Laryngoscopic Image. *Fig. 1.*—In a child aged five. *Fig. 2.*—In an adult male.
Fig. 3.—In an adult female. (In the cadaver.)



Fig. 1.



Fig. 2.

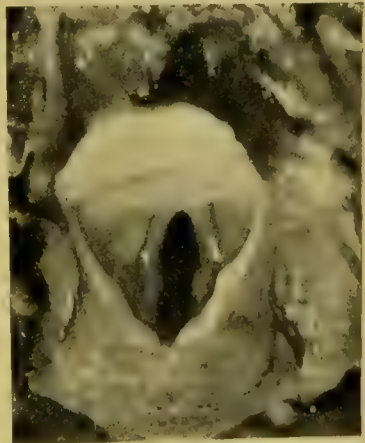


Fig. 3.

vous persons, deep inspiration may be attended with partial adduction of the vocal cords, simulating abductor paresis. By making the patient sound a prolonged note until he involuntarily takes a deep inspiration, the true abductor action is manifested.

Congenital malformations.—I have once seen a persistence of the foetal occlusion of the glottis by a membranous web; it occupied the anterior two-thirds of the glottic aperture. It was observed many years ago in a young child with no suspicion of syphilis, while I was acting as clinical assistant at the Golden Square Throat Hospital. Mackenzie and Poore have recorded similar cases, and Semon has observed a case in which, at the same time, there was a coloboma of both eyes.



FIG. 32.

Partial occlusion of the glottis by remains of foetal web.

The epiglottis may present a deep central notch, even amounting to a bifid epiglottis.

Laryngoscopy in children and infants.—In very young or nervous children a good view of the larynx may not be possible by the ordinary laryngoscopical methods, and for these patients we may have resort to special methods, such as those of Lambert, Lack, Escat, Petersen, or Rauchfuss.

With the child held sitting upright in an attendant's lap, and the head retracted, Lack passes the left forefinger into the mouth, and with the finger-tip well in the patient's right vallecular or pyriform fossa, pulls forward the base of the tongue and with it



FIG. 33.

The distal end of Escat's Laryngoscopic Tongue Depressor.

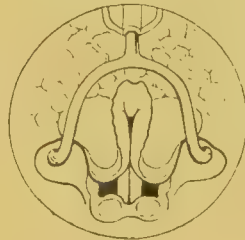


FIG. 34.

To show how the Tongue Depressor should be applied to the base of the tongue with the ends of the fork resting in the sinus pyriformis.

the epiglottis. By this manner one can obtain a complete view of the larynx by means of the laryngoscope used in the usual manner. For children who have cut their teeth he uses a gag, or else substitutes for the finger a flat copper band, like a tongue

depressor, with the last half-inch abruptly bent down at a right-angle with the lingual portion.

Rauchfuss long ago resorted to simple depression of the child's tongue and immediate introduction of a mirror, using a tongue depressor, such as Fränkel's, which, when placed far back on the dorsum, allows of some slight forward traction of the dorsum and epiglottis. Mount Bleyer and Petersen introduced modified tongue spatulæ used in the same manner as Lack's tongue retractor. Escat uses a similar, but heavier, spatula, with the child deeply under chloroform. Kirstein's spatula acts on the same principle as Rauchfuss' instrument, and often is successful in yielding a satisfactory inspection of the posterior half of the larynx.



FIG. 35.
Lack's Tongue Retractor.

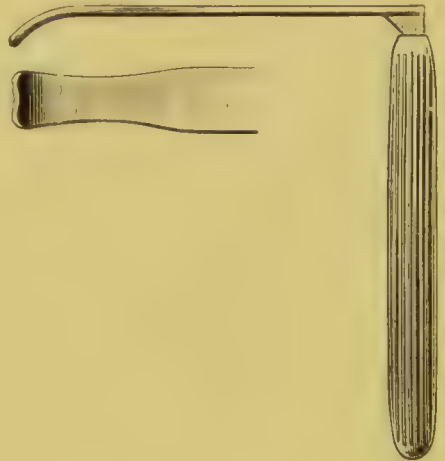


FIG. 36.
Kirstein's Tongue Depressor.

Trans-illumination of the larynx is rarely of any value, but has been of assistance in noting infiltration of the parts by a growth, or in detecting the consistence of a growth; thus, Mackenzie was enabled to diagnose a small laryngeal tumour as cystic by this means. The room being completely darkened, a strong light is concentrated in the front of the larynx externally, with the laryngeal mirror *in situ*, sufficient light being transmitted to illumine the larynx.

Skiagraphy of the larynx may be employed with advantage sometimes in the detection of foreign bodies, which are more or less opaque to the rays. The focus tube is best placed above the patient, who should lie on the side, so that a lateral skiagram

of the larynx, is obtained. It is possible that in the near future this method may prove of service in the differential diagnosis of tuberculous deposits and neoplasms; in fact, Sendziak, in a case of laryngeal paralysis, was enabled, by means of the X-rays, to confirm his suspicion that it was due to compression by enlarged glands, secondary to carcinoma of the œsophagus.

Autoscopy of the larynx.—Kirstein has introduced a special spatula, with the distal end bent down at a right-angle with the main portion of the pressor. By introducing the instrument well back, till the hook-like end lies in the glosso-epiglottic space, he can draw the epiglottis forwards while pressing down the base of the tongue, so that, with the patient in the correct position, an uninterrupted view of the posterior half of the larynx can often be obtained. The patient must sit straight, leaning a little forwards, with the head retracted, the examiner standing in front while he makes firm pressure with the instrument described. Among the advantages claimed by Kirstein for his method are that the parts which can be seen are seen by direct vision, and not by reflection through a mirror, and that laryngeal applications

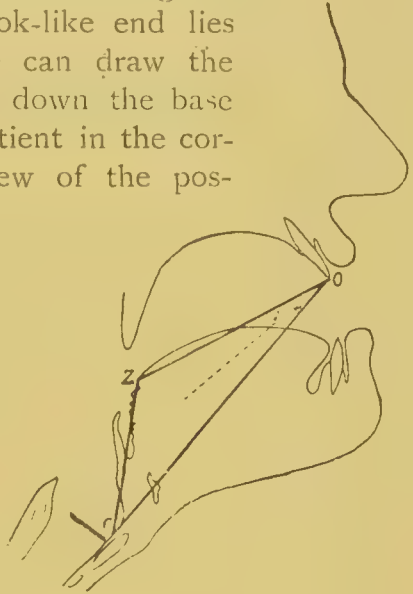


FIG. 37.

Diagram to show the position of the tongue in autoscopy.

or operations are more under control. Unfortunately, it is seldom possible to see more than a limited portion of the posterior half of the larynx, and to one who has become accustomed to the indirect method autoscopy presents so many disadvantages, that its field of usefulness is most restricted.

RHINOSCOPY.

The actual anatomical relations and form of the nasal fossæ have been already described and figured (pp. 1-4, *Plates I, II, XXXIX*, etc). In the living patient they can only be observed "end on" by anterior and posterior rhinoscopy.

The nasal passages may be examined from the front by anterior rhinoscopy, and through the mouth from behind by means of

the rhinoscope—posterior rhinoscopy. The patient and examiner sit facing one another, the light being arranged as in laryngoscopy. In examining the interior of the nose it is necessary to observe not only the presence or absence of pathological conditions within the nasal passages, but also to note any indications of disease involving the accessory sinuses which open into the nasal fossæ. For this purpose a brilliant illumination is even more essential than in laryngoscopy, while the same forehead mirror of about fourteen-inch focus should be used.

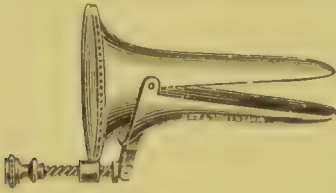


FIG. 38.

Duplay's modification of Bresgen's Speculum.

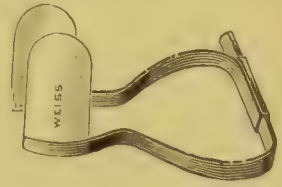


FIG. 39.

Browne's Nasal Speculum.

Anterior Rhinoscopy.—For this we require a nasal speculum. Fränkel's is simple, but the vibrissæ are apt to project through the fenestræ of the blades and obscure the parts beyond, and therefore I think that for general use one of the best is one consisting of two ivory blades on sliding bars, as it may be used in cautery operations as well as for examination purposes. The spring of Thudichum's is liable to cause pain, unless very carefully

held. The most comfortable to the patient is Bresgen's, but it is more difficult to manipulate. Some form of self-retaining speculum, like Cresswell Baber's or Neil-Griffith's, is desirable for operations on the nose requiring two hands. A few fine silver probes, such as Pegler's, to investigate the nature and con-

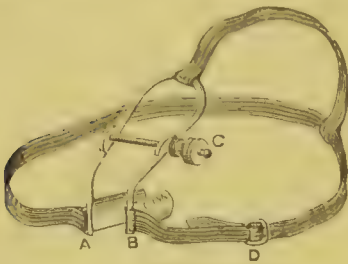


FIG. 40.

Neil-Griffith's Self-retaining Speculum

sistency of the various prominences and swellings encountered complete the apparatus for anterior rhinoscopy.

Having previously inquired for any symptoms noticed by the patient, such as nasal obstruction, discharge, foul odour, loss of smell or taste, pain, sneezing, etc., let the light be focussed on the anterior nares. But before introducing the nasal speculum, raise the tip of the patient's nose and examine the vestibule and the

front part of the nasal passages, otherwise ulcers, septal perforations, or other abnormalities may be subsequently concealed by the blades of the speculum and escape detection.

Then, with the patient's head very slightly retracted, insert the speculum and gently separate the blades, directing the light well into the passage. Observe the olfactory fissure, and note its width and whether there are any collections of abnormal secretion. The inner wall is formed by the septum, covered with yellowish pink mucous membrane; any departure from the normal contour, the presence of ulceration, new growths, septal deviations or perforation will be easily recognised here.

On inspecting the outer wall, the inward projection of the inferior turbinated body first arrests attention. Even when healthy it varies considerably in colour and size, according to the state of the erectile tissue; for when the venous sinuses are distended the turbinal body appears as a red or pink, tense swelling, as shown in *Plate XVIII, Fig. 2*; but when collapsed it is pale pink, and is much less prominent. The turbinals are shown in this figure in a state of distension.

By directing the patient to throw the head well back so that the light is directed towards the roof of the nose, we bring into view the anterior portion of the middle turbinated body, which is similar to the inferior in colour and consistence. In a very few patients only can the superior turbinated body be made out, far back and high up. The presence of polypi in the middle meatus, or any discharge of pus here, should lead to a careful

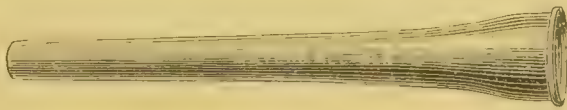


FIG. 47.

Zaufal's Funnel Speculum for examining the posterior wall of the rhino-pharynx.

investigation of the region of the *hiatus*, while the existence of any growth, ulcerated surface, collections of crusts or secretion, spurs or deflections of the septum will be observed. Seldom can we see more than the anterior border of the superior turbinated body. Sometimes it is possible to see the posterior pharyngeal wall through the normal nasal passages, especially when the olfactory fissure is wide, and then, on swallowing, the salpingo-palatine fold is seen crossing inwards.

The presence or loss of sensibility, or the existence of hyperæsthetic spots, may be determined by touching the lining mucous membrane with a probe. The nasal probe is also employed to investigate the consistence of any growth, and whether it is fixed or movable; vascular engorgement of the turbinal bodies gives the sensation of a cyst distended with fluid, and pits on pressure. By spraying the parts with a 5 per cent. solution of cocaine, the vessels are emptied, and the swelling due to simple vascular distension disappears, and thus not only do we get a clearer view of the parts beyond, but we can determine how much of any swelling is due to vascular engorgement, and how much to true hyperplasia of the tissues. A fresh solution (2 to 10 per cent. of supra-renal extract or powder is a more potent astringent than cocaine, and it is sometimes desirable to use it alone, or after cocaine, to obtain a more perfect inspection of a narrow nasal passage.

Posterior Rhinoscopy is more difficult of accomplishment, and requires considerable practice before a satisfactory examination can be made. One of the smaller laryngeal mirrors (half inch diameter) may be used for the purpose, but a special form of rhinoscopic mirror, such as Fränkel's or Michel's, with a movable mirror, is much easier to manipulate.



FIG. 42.
Michel's Rhinoscope.

Direct the patient to open the mouth without protruding the tongue, the head being erect, then with the left hand depress the dorsum of the tongue. For this purpose either Fränkel's or Türk's depressor is very convenient, as the form of the handle enables the left hand to be kept well out of the way of the right which holds the rhinoscope. In using it care must be taken to place it just beyond the dorsum of the tongue, and no further; for if not far enough back the tongue bulges up and occludes the view, while if too far back gagging and retching are induced.

A successful examination can only be obtained by carefully avoiding any titillation of the fauces on introducing the mirror

and by setting the patient completely at ease. I generally find it is better to give no directions as to the manner of breathing, but simply to let the patient breathe quietly, and generally the soft palate will very soon relax spontaneously. If it does not

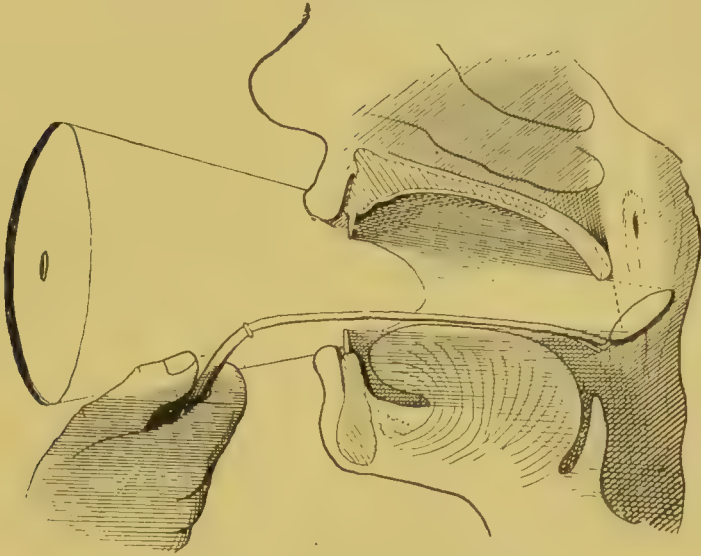


FIG. 43.

Posterior rhinoscopy (Carwardine). (From the author's article in "Carwardine's Practical Surgery.")

do so, instruct the patient to *breathe in* through the nose. If this manœuvre fails, we must use a palate retractor to draw forward the soft palate. Voltolini's or White's self-retaining retractors are convenient forms. Before using it, the fauces and the back of the soft palate should be cocainised, and sometimes the application of cocaine obviates the necessity for the retractor. As soon as the soft palate ceases to occlude the naso-pharynx, the rhinoscope should be introduced and passed below and well



FIG. 44.

Voltolini's Palate Retractor.

behind the velum, care being taken to avoid touching the fauces or pharyngeal wall.

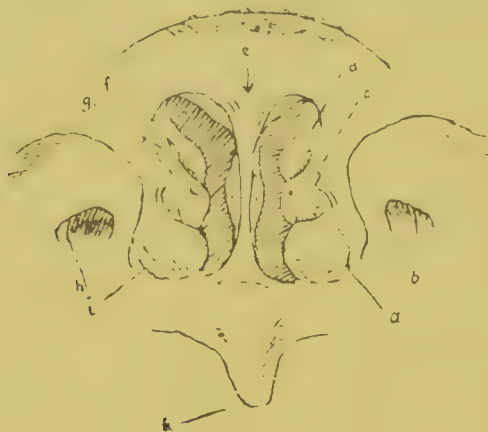
First, by gradually tilting forward the mirror till it assumes an angle of about 45° , as shown in the diagram (*Fig. 43*), the posterior border of the septum nasi is brought into view in the centre of the image; it forms a convenient landmark (*see Plate*

IX, Fig. 6). Then on slightly turning the mirror, first to one side, then to the other, the *choanae* or posterior openings of the nasal passages come into the field, and, projecting inward, the posterior extremities of the inferior and middle turbinal bodies appear as "frogspawn-like," grayish-white bodies. Owing to the very oblique direction in which the parts are viewed in



FIG. 45.
Cresswell Baber's modification of White's self-retaining Palate Retractor.

rhinoscopy, the lower turbinals appear to rest on the upper surface of the soft palate, the inferior meatus being seldom seen at all. Sometimes the superior turbinal can be dimly seen in the upper part of the choanae. Below, the superior or posterior surface of the soft palate and uvula come into view, and, turning the mirror to either side, we can see the orifices of the Eustachian



Patient's right. Patient's left.

FIG. 46.

Explanatory Diagram of the Rhinoscopic image, *Plate IX, Fig. 6*. *e*, the septum, on either side of which are the *choanae narium*; *a, c, d*, the left inferior, middle and superior turbinated bodies; *i, g, f*, the right ditto; *b* and *h*, the left and right Eustachian tubes; *k*, posterior surface of the uvula.

tubes, and behind them the fossæ of Rosenmüller. Finally, after once more finding the posterior border of the septum, which acts as a convenient landmark, the roof and posterior wall of the rhino-pharynx are successively brought into view, as the mirror becomes more and more horizontal before it is withdrawn.

Of course only small portions of the rhino-pharynx can be seen in the rhinoscopic mirror at any one time, but by mentally connecting the several successive images we obtain a complete rhinoscopic image as depicted on the plate.

While making the examination, the presence of any growth or collections of secretion, or other abnormalities should be carefully noted. The presence of a certain amount of lymphoid tissue in the vault and posterior wall is normal in children up to the age of about 16 to 20. In some cases this pharyngeal tonsil shows the "recessus medius" very plainly, or even a short tubular recess.

It is scarcely necessary to add that the rhinoscopic examination must be very rapid, and very often we have to make several short successive examinations, or even be satisfied with such a momentary glance that only a practised eye could see the condition of the parts.

Congenital Defects and Deformities.—We need not here refer



FIG. 47.
Case of Malformation of the Nose (W. R. H. Stewart).

to various external congenital defects of the nose, conditions which come under the scope of general surgical text-books,

excepting that central depression at the tip of the nose which is analogous to hare-lip or cleft-palate.

It is not very unusual to find the anterior orifice of the nasal passages partially obstructed by a web-like band projecting up from the floor of the vestibule about half an inch from the margins of the alæ. Very rarely this web of skin completely occludes the nasal passages, representing the persistence of a condition analogous to the congenital web sometimes observed partially occluding the glottis. I have seen more than one such case, another is recorded by Jarvis.

A congenital osseous occlusion of the posterior nares, either unilateral or bilateral, has been observed, but such conditions are extremely rare.

The most frequently occurring congenital defect is deviation of the septum. It is, in fact, unusual to find a septum which is perfectly straight. The condition is referred to more fully under the head of deviated septum. The septum has been found to be doubled (splitting of the septum), and very rarely the septum is continued back into the naso-pharynx, which is then divided into two chambers more or less completely.

The developmental origin of these congenital defects is described in the section on embryology.

EXAMINATION OF THE EAR.

Owing to the intimate anatomical connection between the middle ear and the rhino-pharynx, the ear becomes secondarily affected in a large percentage of patients with nose and throat diseases, and in children deafness is usually due to abnormal conditions of the nose or throat. Hence it is very often essential that the examination of the patient should include investigation of the nature and cause of ear defects, so that one may determine how far the aural complication depends on and may be relieved by treatment of the upper respiratory tract.

The organ of hearing is anatomically subdivided into three parts, (1,) the external ear, consisting of the auricle and the external auditory meatus; (2,) the middle ear or tympanum, comprising the tympanic cavity and its contents, the mastoid antrum and cells, and the Eustachian tube; and (3,) the internal ear, including the osseous and membranous labyrinth, and the endings of the auditory nerve in the cochlea and vestibule.

The intimate anatomical connection of the tympanic cavity, and through it the mastoid antrum, with the rhino-pharynx, are well shown in *Plate XVII*; and the anatomy of the various structures in the middle ear in *Plates XXXVI* and *XXXVII*.

Equality of air-pressure on both sides of the tympanum is maintained by the opening of the pharyngeal orifice of the Eustachian tube during swallowing by the action of the levator palati and salpingo-pharyngeus muscles; thus any condition resulting in obstruction in the Eustachian tube or in paralysis of the muscles which open the orifice, which interferes with the entrance of air through the tube, causes the drum to be compressed as the intra-tympanic air becomes absorbed. Catarrhal affections in the nasal passage or rhino-pharynx are prone to spread to the Eustachian tube, which also affords a channel through which various micro-organisms may readily spread to the tympanum and mastoid antrum.

The chief symptoms requiring investigation in connection with affections of the ear are:—

(1.) Defects of hearing; (2.) subjective noises, tinnitus aurium.

(1.) DEFECTS OF HEARING.—Deafness is termed *obstructive deafness* when due to affections of the sound conducting structures, *i.e.*, in the external or middle ear, and *nerve deafness* when the lesions are in the sound perceiving portion, *i.e.*, the labyrinthine and intracranial distribution of the auditory nerve. The degree of deafness is estimated by means of various tests, *viz.*, the voice, a watch, tuning forks, and Dalton's whistle.

For the VOICE TEST the ordinary conversational voice and whispering are employed. The opposite ear being closed the examiner speaks some words at some distance from the patient, standing well to the side of the ear to be tested, and while repeating the word he approaches the ear till the patient can hear it, the distance being measured.

The same method applies to the whispering test. If neither whispering nor the ordinary conversational voice can be heard, the "loud" or "shouting" voice test should be tried. The result in all cases is noted for each side in feet or inches.

The WATCH TEST is employed in the same manner, but in all cases the watch should be placed out of hearing of the patient

first and gradually brought up to the ear till it is heard. The result is noted in the form of a fraction, the numerator being the number of inches from the ear at which the patient hears it, and the denominator the number of inches at which the normal ear should hear it (previously ascertained by testing). Thus, if the tick of a particular watch is heard normally at 40 inches, and the patient generally heard it at 12 inches distance, the condition is noted as $\frac{12}{40}$ inches.

THE TUNING-FORK TEST.—The “voice” and “watch” tests are used to estimate “air conduction,” the conduction of sound by the air to the tympanum and thence by the ossicles to the fenestrum ovale. The tuning-fork is used to estimate both “air-conduction” and “bone-conduction,” *i.e.*, the conveyance of vibrations through the mastoid to the labyrinth. The tuning fork should not be of a high pitch, and should be so short that

the vibrations soon become too weak to be heard. Gardiner Browne's, tuned to the middle C, is a useful form. For testing air-conduction it is held, while vibrating, close to the external meatus, without touching the ear, and for bone-conduction the button or foot is held pressed gently against the mastoid.

Swabach's Test.—The vibrating fork is first held to the patient's ear, who is directed to say at once when the sound “stops;” it is then held to the examiner's ear (presumably normal) and he notes how many seconds longer he can hear it, thus indicating the difference between the number of seconds it is heard by the patient's deaf ear and the examiner's normal ear. The number of seconds is noted as a minus quantity. This is done for each ear separately.



FIG. 48.
Tuning fork.

The same process is followed for estimating “bone-conduction,” excepting that the tuning fork is held first to the examiner's mastoid

and then, the moment the examiner ceases to hear it, to the patient's mastoid.

If the patient fails to hear the tuning fork when the examiner just ceases to hear it, his bone conduction is not increased, and

the test is again applied, this time the tuning fork being first applied to the patient's mastoid and then to the examiner's. It may then be found that the examiner hears it longer than the patient; in other words the bone conduction is "diminished," instead of being "normal" or "increased." Increased, diminished, or normal air and bone conduction may be indicated respectively by the signs + — or =, while for inability to hear any sound the sign 0 may be used, the difference in seconds between the patient's hearing power and the examiner's being indicated by numbers.

Thus if the patient hears the tuning fork on the mastoid for ten seconds longer than the examiner and for fifteen seconds less when it is held to the meatus, we write:—

$$\text{T. F.} = \frac{\text{mastoid} + 10}{\text{meatus} - 15}$$

Rinné's Test.—In a normal person the tuning fork ceases to be heard sooner when held on the mastoid than when held to the meatus, *i.e.*, air conduction is better than bone conduction. Rinné's test is then said to be positive (Rinné +), but if the bone conduction is better than the air conduction, Rinné's test is negative (Rinné —).

If the tuning fork after it has ceased to be heard at the meatus is heard for seven seconds on the mastoid, air-conduction is less than bone-conduction by seven seconds, *i.e.*, air < 7 bone. If the air conduction is — 10 and the bone conduction + 15, when compared with the normal: write:—

$$\text{T. F.} = \overset{-10}{\text{Air}} < \overset{+15}{7 \text{ Bone}}$$

Interpretation of Swabach's and Rinné's Tests.—Where bone conduction is increased and air conduction diminished, as compared with the normal ear, the deafness is due to middle ear disease. But, if on the contrary both air and bone conduction is diminished, deafness is due to labyrinthine disease (that is, provided there is no obstruction, *e.g.*, by cerumen, in the external meatus).

Rinné + generally indicates absence of middle-ear disease.

Rinné — generally indicates some disease of the middle-ear, and in the absence of abnormal conditions (*e.g.*, a plug of cerumen) in the external ear, points to nerve deafness.

But Rinné — only indicates middle-ear disease if the tuning

fork be low pitched. Moreover it cannot be relied upon as a proof of middle-ear disease (*a*,) in very high degrees of deafness ; (*b*,) in old persons in whom the difference between air and bone conduction is normally diminished.

WEBER'S TEST.—A vibrating tuning fork is placed on the forehead or vertex. If heard better in the worse ear, the lesion is in the conducting apparatus, and Weber is said to be + ; if heard better in the better ear Weber is termed —.

GALTON'S WHISTLE AND GRADUATED TUNING FORKS are used to test the hearing power for sounds of varying pitch, "range of tone hearing." In using Galton's whistle it should be held about a foot and a half from the ear and the ball pressed gently so as not to produce too loud a sound or create too much "rushing of wind" sound. When the loss of hearing is most marked for high tones—the low tones being better heard—labyrinthine or nerve deafness is indicated : conversely impairment of hearing most marked for low tones is generally due to middle ear disease.

Paracusis Willisii is the term applied to the anomaly of hearing better in a noise, *e.g.*, when conversation is better heard in a train, and when present the deafness is almost certainly due to middle ear disease. On the other hand, when the deafness is more marked in the midst of noise, or when the patient is tired, it is usually a case of nerve deafness.

Diplotacusis or double hearing for speech or tones, or for both, is probably due to derangement of the harmonious action of the right and left cochlea.

Hyperacusis or *Hyperæsthesia acustica*, or the exaggerated, and often painful, perception of individual tones or of all sounds, is due to the auditory nerve being in a state of irritability : it is sometimes observed in hysterical patients.

(2,) *TINNITUS AURIUM*, or the perception of subjective noises, may be continuous or intermittent. If continuous, described as a hissing, singing, humming, or rushing sound, or like an engine blowing off steam, it is probably due to increased tension in the middle ear, or to anæmia or venous congestion. The first class will be relieved by Politzerisation, the second will be relieved on lying down and worse on getting up, while if due to venous congestion the noises will be aggravated by stooping or lying down.

Intermittent or pulsating tinnitus is due to vascular engorgement either in the middle ear or in the labyrinth. Dundas Grant has shown that these two classes of cases may be differentiated owing to the fact that the vascular supply in the middle and external ear is derived from the territory of the carotid and its branches, while the internal auditory arteries come from the basilar artery which is formed by the junction of the two vertebral arteries. Hence if compression of the carotid artery diminishes or checks the tinnitus in the corresponding ear, the congestion is in the middle or external ear; whereas if compression of both vertebral arteries in the sub-occipital triangles diminishes or checks the tinnitus, the congestion is labyrinthine.

OBJECTIVE EXAMINATION.

Examination with the aural speculum.—For a satisfactory inspection bright daylight or artificial light is necessary, with the same reflecting mirror as used in laryngoscopy. Firstly, attention should be directed to the external ear and the outer portion of the external auditory meatus, and any redness, swelling, or discharge noted; especially should any tumefaction or tenderness over the mastoid antrum be carefully observed. The upper part of the pinna should then be grasped and drawn somewhat upwards and backwards so as to render the auditory canal as



FIG. 49

The normal right membrana tympani.

(1) The membrana; (2) The handle of the malleus, ending in the umbo; (3) short process; (4) the posterior and (5) the anterior fold; (6) membrana flaccida; (7) the bright spot.



FIG. 50.

The appearance presented by a depressed (right) membrana tympani. The handle of the malleus is foreshortened and the short process very prominent. The posterior fold is abnormally prominent. The bright spot is more diffuse and less distinct towards the periphery of the membrane.

straight as possible, and a suitable sized aural speculum insinuated with rotary movement, as far as the bony meatus. By slightly varying the direction of the speculum the various portions of the membrana tympani can then be inspected until the whole has been clearly brought to view. The meatus may be blocked by cerumen, discharge, foreign bodies, polypi, or by simple swelling

of the lining tissues. Should cerumen or discharge block the canal or obstruct the view of the membrana, the speculum should be withdrawn and the obstructing matter either syringed out with warm water or removed by pledgets of cotton wool on a probe.

The normal tympanic membrane is pinkish pearl-gray in colour, with polished surface, concave in shape, and placed obliquely, so that the light reflex forms a triangular bright spot on its lower part. The handle of the malleus can be seen through the translucent membrane traversing the upper half obliquely from above downwards and backwards. At the upper end of the handle of the malleus the knob-like projection of the short process may be seen. Above this process, in the notch of Rivinius, is the *membrana flaccida*, or Schrapnell's membrane covering Prussak's space and bounded below by two folds of membrane, the *anterior and posterior folds*, which pass respectively forwards and backwards from the short process towards the periphery of the *membrana tensa*.

As regards the ear-drum, the main points to note are: (1,) its colour; (2,) its mobility; (3,) the presence or absence of retraction; (4,) the existence of perforations.

The mobility of the tympanic membrane may be tested (1,) by Siegle's speculum; 2, by inflation of the Eustachian tube by Valsalva's method.

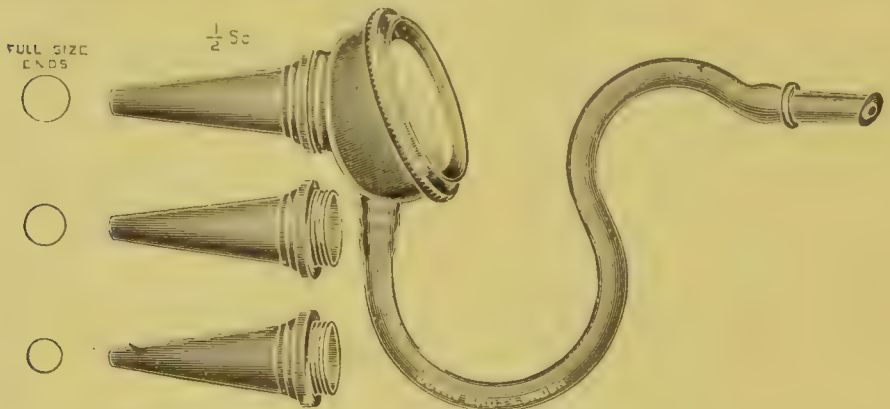


FIG. 51.—Siegle's Speculum.

Siegle's speculum is practically a ear-speculum, to which is fitted an air-tight chamber covered with a glass lens and connected by a tube with a ball or mouthpiece, by means of which the air within may be exhausted or compressed, while the membrane is being inspected through the glass cover. In using it, it is of

course essential that the end should very accurately fit the external auditory meatus.

Retraction of the membrane, which may be general or circumscribed is usually the result of obstruction of the Eustachian tube from catarrh. The appearance is very characteristic, the handle of the malleus is foreshortened, the short process sticks out, and the anterior and posterior folds are unduly prominent. The light spot loses its triangular cone shape and becomes diffuse.

Inflation of the Eustachian tube.—The three methods commonly employed are: (1,) Valsalva's method; (2,) Politzer's method; (3,) Inflation through the Eustachian catheter.

Valsalva's method.—Direct the patient to hold the nose firmly, compressing the *alæ nasi* with the forefinger and thumb, and then to expire forcibly at the same time puffing out the cheeks. If the Eustachian tubes are pervious this manœuvre will sometimes succeed in driving the air through them so as to distend the ear-drums: but it does not follow that there is any Eustachian obstruction if the tubes cannot be inflated in this manner.

Poltzer's method.—This valuable diagnostic and therapeutic method is based on the fact that during deglutition the pharyngeal orifices of the Eustachian tubes are opened by the action of the salpingo-pharyngeus muscles, so that if air be then driven in through the nasal passages, it is forced up the open orifice of the Eustachian canal.

This action of the salpingo-pharyngeus muscle is the physiological means of adjusting the air-tension within the tympanic cavity to the atmospheric pressure, and Politzer's method takes advantage of this normal action. In employing the method, the patient being seated, takes a *small* sip of water in the mouth and is directed to hold it until he is told to "swallow." The examiner then inserts the nozzle of a Politzer's inflation bag into one nostril, while he closes the other nostril by firmly compressing the nose between the finger and thumb—then on directing the patient to swallow he immediately compresses the bag, driving the air into the nostril. If the manœuvre has been successful in inflating the Eustachian tube the patient experiences a dull thud in the ear, and the ear-drum remains over distended, until by swallowing again the excess of air escapes once more from the middle ear. But the procedure is additionally valuable if, prior to the inflation of the ear, a rubber auscultation tube,

with suitably shaped ends to fit the ears, connects the ear of the patient to one ear of the examiner. A little practice will enable the practitioner to hear for himself, through the auscultation tube, whether the patient's Eustachian tube has been inflated, and will also reveal by the sounds or sensations produced in the examiner's ear, whether a perforation exists in the inflated ear. Large perforations yield a peculiar low-pitched rushing sound, small perforations, a squeaky or high-pitched sound.

While in many cases both of the patient's ears will be inflated by Politzerisation through either one of his nostrils, the ear corresponding to the inflated nostril is most inflated, and often is the only one inflated. So that one should always begin by Politzerisation through the nostril of that side which corresponds to the ear we desire to affect.

Failure to inflate may depend firstly, from the patient swallowing too soon or too late, in which case the manœuvre should be repeated ; or, secondly, owing to the Eustachian tube being too much obstructed. The latter difficulty may sometimes be overcome by directing the patient, after taking the sip of water, to tilt the head well over towards the shoulder of the side of the ear which we wish to inflate.

It is well to begin by somewhat gentle inflation lest, with a very permeable Eustachian tube, the membrana be distended with such abruptness and force as to cause grave discomfort or pain ; it is always easy to increase the inflating force subsequently if necessary.

Catheterisation of the Eustachian tubes.—This method consists in passing a Eustachian catheter along the floor of the nose into the orifice of the Eustachian tube, and then inflating the tube by means of a Politzer bag attached to the Eustachian catheter while *in situ*.

The examiner stands on the patient's right hand, and the patient should be seated upright in a high-backed chair with the head resting against the chair or otherwise steadied. It is well to spray the nasal passage with a 4 per cent. solution of cocaine. When sensation is dulled, a Eustachian catheter of suitable calibre and curve, held lightly between the finger and thumb, with the curved end turned down, should be cautiously passed horizontally backwards along the floor of the nose, until it impinges against the posterior wall of the pharynx. The

catheter is now turned inwards, through one quarter of a circle, so that the curved nozzle looks horizontally inwards, and then withdrawn for about half-an-inch until it is arrested against the posterior margin of the nasal septum. Then rotating the catheter outwards through three-fourths of a circle, the end should slip into the Eustachian orifice. Holding it gently but firmly and very steady, as it lies in the Eustachian orifice, the Politzer bag is inserted into the end of the Eustachian catheter outside the nose, and the tube inflated. Either the patient's sensation, or the more certain method of the auscultation tube already mentioned, indicates whether the ear has been inflated or whether the Eustachian catheter has failed to enter the Eustachian orifice. In the latter event the Politzer bag should be detached and the process of finding the Eustachian orifice gone through afresh (without removing the catheter from the nose altogether) until success has been attained.

It is desirable to use the largest size catheter that will readily pass through the nose, since the larger ends are less uncomfortable for the patient's Eustachian tubes. If the curve of the catheter end is too considerable to allow its passing along the nasal passage, it may be straightened a little, or a shorter portion of the extremity made to take the required curve.

Beginners often fail owing to the end of the catheter slipping either into the fossa of Rosenmüller or in front of the Eustachian lips, when it is rotated.

If the hearing power as tested by the watch or voice tests, manifestly improves on Politzerisation or the passage of the Eustachian catheter, it affords strong evidence of obstructive deafness due to catarrhal or other affections spreading up from the throat. The nasal and throat affections in which the ear is prone to be secondarily affected, and the indications for their removal, are described elsewhere in connection with these various diseases.

GENERAL SEMEIOLOGY.

Knowledge of any special branch of medicine carries special dangers unless it be tempered by good judgment: thus in studying the manifestations of disease in any one region of the body it is always essential to guard against narrow views and the undue estimation of local symptoms or abnormalities. Therefore, before passing on to the detailed description of the nose

pharynx and larynx, and their diseases, it may be helpful to briefly review some general constitutional symptoms associated with disease in these regions, in order to direct attention to the very important part that general therapeutical measures should occupy in their treatment, and to emphasise the desirability of examining the pulse, heart, lungs, etc., and of investigating the personal and family history of patients complaining of their throat and nose, so as to avoid the error of having regard only to local manifestations of general affections.

Gout, rheumatism, and dyspepsia, are very frequent causes of pharyngitis and laryngitis, and of certain forms of rhinitis; and though from long-standing congestion local treatment may be required, the great majority of cases yield only to appropriate general treatment.

As an instance of the importance of taking into consideration the general condition of a patient's health, I may cite the case of a clergyman sent to me by his medical attendant for painful sore throat and loss of voice. He was the Vicar of a large country parish, and was thoroughly overworked by his Lenten services. The fauces were intensely injected, and he complained of severe pain, especially in swallowing. The larynx was greatly congested, and he was almost voiceless.

But on feeling his pulse, I found that it was frequent, 96, small and irregular, while the heart beats were 136 to the minute! There was no history of gout or rheumatism, yet the patchy appearance of the faucial and laryngeal congestion confirmed my suspicions that an indefinite swelling about one elbow joint was gouty. He rapidly improved under the appropriate general treatment which I was led to suggest to his medical attendant, and with the help of a sedative spray and pastil, he was very soon restored to fair health. It is scarcely necessary to point out that to have had regard only to the throat condition in a patient whose heart action was so weak and feeble that nearly every other beat failed to reach the wrist, would have been disastrous.

Again, a slight persistent laryngitis may be the earliest symptom of tubercular disease of the lungs, and should lead us to examine the chest and feel the pulse.

Paralysis of a vocal cord may be caused by pressure of an aneurysm on a recurrent laryngeal nerve, and is often the earliest indication of an intra-thoracic tumour, or it may be due to some

central nerve lesion, such as locomotor ataxia, bulbar paralysis, nuclear disease from syphilis, or to an intracranial growth.

Intra-nasal disease is the exciting factor in a large percentage of spasmodic neuroses, *e.g.*, hay-fever, persistent cough, asthma, or even epilepsy, migraine, etc. On the other hand, epistaxis may be a symptom of typhoid fever, influenza, chronic renal disease and other general affections.

Patients with throat affections generally complain either of pain, impairment of voice, respiratory obstruction, difficulty in swallowing, or increased secretion.

Pain may be very considerable in simple dyspeptic or gouty pharyngitis, in malignant disease, and in tuberculosis, but, on the other hand, it is often almost absent in early malignant disease, and is usually slight in syphilis and lupus.

Vocal impairment may be due to thickening of the pharyngeal mucous membrane, or perforation of the velum palati, or the voice may be nasal in character from nasal obstruction. The cause of vocal alteration may be in the larynx and amount to hoarseness, or loss of voice (*aphonia*), and may arise from ulceration, inflammatory thickening of the vocal cords, imperfect approximation of the cords from growths, thickening in the inter-arytenoid fold, or from paresis of the vocal cords. Not infrequently hoarseness is the *only* symptom noticed by the patient in commencing laryngeal cancer or in intra-thoracic aneurysm. It acquires a peculiar raucous character in old syphilitic disease, which is almost pathognomonic.

On the other hand, the voice may be quite unaltered in the most dangerous form of vocal cord paralysis, *viz.*, bilateral paralysis of the abductors, and hardly appreciably altered in paralysis of one vocal cord.

Obstruction to respiration results from nasal obstruction in rhinitis, polypus, growths or deviation of the septum; and when the respiratory difficulty lies in the nasal passages, it is sure to cause various pathological conditions in the rhino-pharynx, if not also in the larynx. Nasal respiration must be carefully investigated, for in incomplete obstruction, the respiration may appear to be conducted normally so long as the patient remains at rest, and thus even considerable degrees of nasal obstruction may escape notice. Deposits of false membrane, laryngeal growths, œdema of the larynx, or perichondrial thickening,

may encroach on the glottic space, and give rise to very marked dyspnœa, which may be absent, if only the epiglottis, or the epiglottic folds, are involved. Dyspnœa, of course, results from spasm of the glottis. The only paralysis of the vocal cords which would give rise to great dyspnœa is bilateral paralysis of the abductors, an extremely rare affection.

The obstruction may be infraglottic, and due to infraglottic growths, or œdema, or to a foreign body, or to pressure on the trachea by a neighbouring growth or aneurysm.

Deglutition may be difficult, *dysphagia* ; painful, *odynphagia* ; or absolutely impossible, *aphagia*.

Dysphagia may arise from paresis of the constrictors, as in bulbar paralysis, from obstruction from a growth in the pharynx or œsophagus, from pressure of a growth or aneurysm on the pharynx or œsophagus, or it may be due to a cicatricial stenosis or to a spasm.

Odynphagia is commonly experienced in gouty, rheumatic, and dyspeptic pharyngitis, and in malignant disease; it is generally present in tubercular disease of the pharynx, epiglottis or arytenoid regions, and is usually considerable in perichondritis of the cricoid cartilage.

Aphagia results from complete obstruction of the laryngo-pharynx or œsophagus.

Increased secretion from the fauces and larynx is common to all catarrhal affections, malignant disease, tuberculosis, and sometimes to a less extent in syphilis.

Nasal discharge is increased and is generally muco-purulent in all forms of rhinitis, except the atrophic form, and is also seen in hay-fever, vaso-motor rhinitis, polypus, and in syphilitic, tubercular and lupous disease. A discharge of pus from the nose may come from one of the accessory sinuses.

If fetid, it is probably due to syphilis, ozæna, malignant disease, or antral empyema. The patient is unconscious of the fœtor in atrophic rhinitis, and may not perceive the smell when syphilitic necrosis is present, but the fœtid odour of antral empyema is perceived by the patient himself.

The well known physiognomy of a patient with syphilitic disease which has involved the bony structures hardly requires description here, and the peculiar facial appearance of a child with post-nasal adenoids is equally characteristic.

CHAPTER III.

ACUTE AND CHRONIC PHARYNGITIS.

ACUTE CATARRHAL PHARYNGITIS—ACUTE SEPTIC INFLAMMATION OF
THE PHARYNX AND LARYNX—RETRO-PHARYNGEAL ABSCESS.

CHRONIC CATARRHAL, HYPERTROPHIC, AND ATROPHIC PHARYNGITIS—
HÆMORRHAGES FROM THE PHARYNX AND LARYNX.

ACUTE CATARRHAL PHARYNGITIS.

Etiology.—The causes of the affection are :—

(1,) *Idiopathic*—associated with sudden exposure to changes of temperature, *e.g.*, in going into the open air from close and over-heated rooms, and especially prone to occur in damp weather. In this, as in all catarrhal affections of the pharynx and respiratory tract, mouth breathing from nasal obstruction is a frequent and potent predisposing factor.

(2,) *Diathetic*—especially gouty and rheumatic conditions.—Many cases of so-called catarrhal pharyngitis following exposure to damp cold really belong to this class.

(3,) *Toxic*—dependent on gouty or rheumatic conditions, or the action of various drugs, *e.g.*, antimony, mercury, iodide of potassium, and the symptomatic pharyngitis of various exanthemata, such as measles, small-pox, scarlatina, typhoid and typhus fever.

(4,) *Traumatic*—from external violence, or due to injury by a spicule of bone, in swallowing hot fluids, or breathing intensely irritating vapours.

Pathology.—Acute pharyngitis, like acute bronchitis, is generally attributed to a chill, and no doubt the majority of cases follow exposure to damp, cold, and sudden changes of temperature ; but it is not very clear how these causes act. It is certain that those who constantly lead an out-of-door life, and even those who are most constantly exposed to cold and inclement weather, are the least subject to catarrhal affections of the respi-

ratory tract, whereas living in close and over-heated rooms increases the liability to them. Yet the fact that acute pharyngitis, like nasal catarrh, is frequently epidemic and mildly contagious, especially in the spring and autumn, points to a microbial origin, and recent investigations, such as those of W. H. Park, seem to show that staphylococci and streptococci, which are always present in the healthy mouth, increase greatly in number and virulence in damp weather and winter months, and are then capable of setting up acute pharyngitis if applied to the throat. Similarly we explain why acute tonsillitis and rheumatism, which are closely related and are probably due to microbial infection, are associated with cold and damp. Individual predisposition determines to some extent the nature of the attack. Thus, one individual is constantly subject to cold in the nose, another to cold in the throat, whilst others, after exposure to chill, suffer from cold in the chest. The toxic forms of acute pharyngitis, however, are probably due to bio-chemical changes in the tissues.

Whatsoever the exciting cause of the pharyngitis, the pathological changes in the mucous membrane are the same, and consist first in general hyperæmia and round celled infiltration, with at first diminished secretion from the mucous glands, followed in the course of several hours by more or less abundant discharge of greyish, viscid mucus, which becomes mucopurulent as the case progresses towards recovery. The superficial epithelium is shed, and the cells of the deeper layers proliferate. The inflamed mucosa presents a thickened, red and velvety appearance. As the inflammation subsides, the mucous membrane tends to regain its normal aspect, but in many cases a more or less pronounced sub-acute condition persists and finally passes into the chronic form.

The **Symptoms** of course vary greatly according to the severity of the attack, patients often treating themselves by simple and well-known domestic remedies. If due to a chill, the general symptoms of malaise, aching in the limbs, and so forth, will be experienced. In every case soreness of the fauces on speaking or swallowing will be noticed, with a sensation of stiffness in the parts, rendering speech uncomfortable, and the voice hoarse and quickly tired. In the earlier stages, the secretions being arrested, the throat feels harsh and dry, and often there

is a sensation of a hair or something in the pharynx which cannot be got rid of. After a short time the secretion becomes increased in amount, tenacious and viscid, but is never excessive, thus differing from the condition in acute rhinitis and rhinopharyngitis. The tonsils are usually involved, being red and moderately enlarged, and projecting beyond the pillars of the fauces; often collections of cheesy matter are seen projecting from the follicles, or spreading between the follicles, and occasionally even forming an adherent membrane.

The inflammation is rarely limited to the pharynx, and may extend upwards to the naso-pharynx and nasal passages, or downwards to the larynx and trachea. The uvula too is often considerably enlarged, and in acute uvulitis may become double its size.

Diagnosis.—There is rarely any difficulty in diagnosing acute pharyngitis, but we should bear in mind that diphtheria, scarlet fever, measles and septic pharyngitis may begin with symptoms indistinguishable from those of simple idiopathic pharyngitis, and therefore doubtful cases, particularly in children, should be carefully watched for a time.

Treatment.—In many cases very simple treatment is sufficient to check the attack. Thus, if seen at the onset we may prescribe a hot foot bath with a few tablespoonfuls of mustard on going to bed, or an ordinary hot bath followed by eight or ten grains of Dover's powder. In more acute cases sucking ice is grateful to the patient, and cold compresses may be applied to the front of the neck. The bowels should be always freely moved by saline aperients, and food should be light and bland. Internally we may give small doses of quinine or, for gastric catarrh, a bismuth and euonymin mixture. A rheumatic or gouty diathesis is very often the real cause of the condition, and then suitable internal medication becomes necessary.

When the trachea is involved and coughing painful, the inhalation of tincture of benzoin, or the same with chloroform, or a mustard poultice applied to the chest will afford relief.

Various lozenges are useful, such as chlorate of potash, cubebs and guaiacum, cocaine alone, or combined with menthol in a pastil.

As the local inflammation subsides the parts should be painted with astringent solutions, such as are used in chronic

pharyngitis. A markedly œdematous uvula may be freely scarified in its lower half, but unless absolutely necessary, the uvula should not be ablated when acutely inflamed. Any affection in the nose or naso-pharynx, which predisposes to attacks of pharyngitis by causing nasal obstruction, should be attended to.

ACUTE SEPTIC INFLAMMATION OF THE PHARYNX AND LARYNX

(including ERYSIPELAS, ACUTE INFLAMMATORY ŒDEMA, ACUTE
PHLEGMON).

Etiology and Pathology.—Various micro-organisms, streptococci, staphylococci, etc., are found in septic inflammation of the pharynx and larynx, and phlegmonous cellulitis of the neck (angina Ludovici). Very often different micro-organisms are associated, and no one of these septic inflammatory affections can be shown to be caused by any one particular coccus or bacillus to the exclusion of others. Thus Semon has argued on clinical and bacteriological grounds that all the various forms of acute septic inflammation of the throat should be considered as varying degrees of virulence of one and the same pathological process. In fact the general tendency of recent bacteriological work has been to show that anatomically similar morbid conditions may be caused by different etiological agents, and this is well exemplified in the production of a tubercle with certain definite structure by various bacilli other than the tubercle bacillus of Koch.

It must be conceded that erysipelas is generally caused by the streptococcus pyogenes, but Max Jordan has proved that it may also be produced by the staphylococcus pyogenes aureus; on the other hand, Chantemesse found the streptococcus of erysipelas in a case of angina Ludovici, and Biondi the same coccus in acute phlegmonous laryngitis; while the bacillus coli communis once it enters the tissues, may become a pyogenic micro-organism. Newcomb, after reviewing the literature of angina Ludovici, concludes that it is, in fact, one form of septic sore-throat, distinguishable not etiologically, but anatomically; and Kanthack has related four cases in which he found different micro-organisms producing various stages of the same process; these cases, bacteriologically distinct, were anatomically identical.

These septic inflammations are characterised by their violence and by considerable exudation, either serous, sero-purulent, purulent, or gangrenous according to the degree of intensity of the inflammatory process, differences in degree but not in kind. The exudation may form a fibrinous coagulum or false membrane on the surface of the implicated mucous



FIG. 52.

Superficial septic ulcer—due to streptococcal and staphylococcal infection.

membrane, but this is a more or less accidental variation. These inflammations generally occur in adults, but may attack persons of all ages and both sexes. Those whose health is run down and who are debilitated from any cause, for instance, diabetics, are most liable to attack ; but even those who are in apparently good and robust health may succumb.

Symptoms.—Clinically, five degrees of septic inflammation may be recognised :—

(a,) *Superficial septic inflammation*, e.g., the so-called hospital sore-throat ; (b,) *Membranous septic inflammation*, e.g., some cases of pseudo-diphtheria, scarlatinal diphtheria, etc. ; (c,) *Œdematous inflammation*, e.g., acute œdematous tonsillitis, pharyngitis, epiglottiditis, arytenoiditis, cellulitis of the tissues of the neck, etc. ; (d,) *Phlegmonous or suppurative cellulitis* ; (e,) *Gangrenous inflammation*.

The invasion may be marked by prodromal symptoms such as general malaise, chills and feverishness, but the more severe cases are almost abrupt in onset, being ushered in by rigors and rapid rise of temperature. The mild cases often amount to nothing beyond a slight sore throat and general feverishness and indisposition, but the slighter cases may develop into the graver

form. In the severer cases the temperature usually ranges high, and is often of a hectic or relapsing type, profuse perspiration accompanying the rapid falls in temperature. In other cases, particularly those of an asthenic type, the temperature may never exceed 100° F. Rigors occurring late in the course of the disease generally denote the occurrence of suppuration or complications in the lung, etc. Albuminuria and glycosuria are frequently present.

The first symptoms usually noticed are sudden pain in the throat, and dysphagia often passing rapidly into inability to swallow. The further symptoms will, of course, vary according to the part first attacked. If the tonsils are the part implicated the appearances presented will hardly differ from simple acute tonsillitis, either unilateral or bilateral. When the pharynx is involved, the mucosa becomes intensely hyperæmic, with a bluish tint, and rapidly swells, the uvula often being enormously swollen. Rapid œdema develops in any portion of the larynx that may be attacked, and hoarseness soon followed by aphonia and laryngeal obstruction are usually marked features.

Some of the gravest and most fatal cases begin in the pharynx and suppurate in the course of a few days (Senator's acute infectious phlegmon).

Acute phlegmonous pharyngitis is characterised by its tendency to extend to the submucous tissues, and, burrowing beneath the cervical fascia, to extend to the trachea, the œsophagus and the tissues of the neck, etc., or to spread rapidly to the larynx, producing fatal dyspnœa from œdema of the glottis. Finally consciousness is lost, and death soon follows, without any of the vital viscera being affected. Diffuse purulent infiltration of the deeper parts of the pharyngeal mucosa develops, and this condition extends to the larynx and glands, involving secondarily other structures. By extension to the lungs the disease may set up a low form of pneumonia or pulmonary œdema, and death often results from cardiac failure. Pulmonary gangrene and abscess have been known to follow the throat affection.

I have met with only one of these rapidly fatal cases. J.S., male, aged fifty, was perfectly well till 11 a.m. on May 5th, when he noticed pain in the middle of the chest. At 3 p.m. he saw his doctor, who could find nothing much the

Hæmorrhagic Pharyngitis and Tertiary Syphilis.



FIG. 1.



FIG. 2.

FIG. 1.—Septic pharyngitis, with submucous hæmorrhages.

FIG. 2.—Tertiary syphilis, with perforation of the soft palate.

matter. He had pain and difficulty in swallowing from the first. On admission to the Royal Infirmary he held his neck stiffly, the tissues behind the angle of the jaw and of the hyoid region being somewhat swollen and tender. On examination there was some general hyperæmia of the fauces, pharynx, and larynx, but no œdema, and the œsophageal obstruction was evidently just below the cricoid cartilage. On May 14th five or six ounces of pus were coughed up, and he experienced great relief, but he was so excessively weak that he could hardly be induced to swallow, and in a few hours sank and died. The temperature since admission had fluctuated between 99° and 101° . The post mortem examination revealed the remains of a small sloughy abscess that had formed in the upper part of the œsophagus, but there was little to account for the extremely asthenic and rapidly fatal illness, nor was there anything in his general condition and appearance on admission to lead one to suspect the extreme gravity of the affection he was suffering from.

Ludwig's main points in the diagnosis of submaxillary phlegmon are : (1,) A slight inflammation in the throat, which generally disappears in a day or two ; (2,) A peculiar *wood-like* induration of the connective tissues which does not pit on pressure ; this induration spreads uniformly, and is bounded by a well-defined border of unaffected tissue ; (3,) A hard swelling under the tongue, with a bolster-like swelling along the interior of the lower jaw, of deep red, or bluish colour ; (4,) The glands escape, although the disease attacks, or may commence in, the cellular tissue around them.

Phlegmonous pharyngitis and cervical phlegmon may end in resolution ; generally, however, it ends in gangrenous suppuration, and not seldom death quickly follows from septic intoxication, or from asphyxia from extension to the larynx.

Treatment in these cases must be prompt and energetic, our aim being directed towards controlling the local inflammation, to supporting the patient's strength with light nourishing food, and to combatting symptoms of nervous exhaustion or failure of the heart. *Locally*, sucking ice will tend to moderate the intense inflammation, and iced cloths, frequently changed, may be applied around the upper part of the neck. Massei recommends spraying the larynx with perchloride of mercury solution

(1 in 2000). A spray or local application of cocaine to the pharynx or larynx will tend to relieve the congestion and pain, and to prevent the occurrence of spasm, and possibly a solution of supra-renal extract might reduce the inflammatory swelling.

Bedford Browne has given the details of two remarkable cases in which the free application of sinapisms was followed by immediate relief to the symptoms of laryngeal stenosis. He advises the administration of salicylate of sodium or ammonium.

If symptoms of acute dyspnoea appear imminent, the larynx may be freely scarified in the hope that tracheotomy may be avoided.

We must in all cases be prepared to perform tracheotomy at a moment's notice. Very rapid and fatal œdema of the larynx is likely to occur. Intubation has been recommended for these cases, and it has the considerable advantage of obviating the necessity for incising the mucous membrane. The method of performing intubation is described further on.

When suppuration has commenced in the larynx there appears to be less likelihood of the supervention of asphyxia, but the rapid destruction of the deeper tissues may give rise to fatal hæmorrhage. When the acuter symptoms have passed, an anti-septic insufflation may be desirable.

As regards *general treatment*, the indications are to give as much light nutriment as possible, to support the patient with brandy if necessary, and to watch for any symptoms of heart failure. In all cases it is well to give full doses of tinct. ferri perchlor. and to add digitalis and strychnine if the pulse is failing :—

R	Tinct. ferri perchlor.	-	-	-	℥xxv
	Acid. phos. dil.	-	-	-	℥v
	Tinct. digitalis	-	-	-	℥v
	Aq. dest. ad	-	-	-	ʒj

To be taken every four hours.

Quinine seems to control the inflammatory process to some extent, provided the doses are sufficiently large. Four to six grains should be given every four hours for the first few days.

Antistreptococcic serum, if given, should be given early. I am unable to express any opinion as to its value in these cases. It has not proved so valuable in septic affections as was anticipated, but this may be due to the fact that it is generally given

too late. Even diphtheritic antitoxic serum would be of slight value if only used when general infection by the bacillus diphtheriæ had occurred.

When the temperature is high, or if rigors occur, ten grains of phenacetin may be ordered, but great care must be observed not to administer antipyretics in doses which will depress the heart, and unless the temperature be over 101° F. it is better to avoid such remedies altogether.

RETRO-PHARYNGEAL ABSCESS.

Etiology and Pathology.—Suppuration in the cellular tissue of the posterior pharyngeal wall may be primary or secondary. The great majority of cases occur in young children, and are primary or idiopathic, and due to inflammation in the lymphoid tissue of the pharynx, arising from no obvious cause, though predisposed to by rickets or the strumous diathesis. Probably a good many cases are septic in origin, especially when arising in adults. Occasionally it is secondary to a chronic nasal or aural affection left by a former attack of measles or scarlatina. It may be due to caries, generally tuberculous, of the upper cervical vertebræ, or caused by injury from a foreign body.

Symptoms.—The onset may be acute or chronic. If acute, the temperature is raised; the symptoms in young children are easily mistaken for those of croup, but in retro-pharyngeal abscess deglutition, as well as respiration, is rendered difficult. The child's cry has a peculiar throaty sound, and respiration may be very much embarrassed, with a croupy cough and dyspnœa.

Fixation of the head is generally a marked feature, and the glands behind the angle of the jaw on the side corresponding to the abscess become enlarged, indurated and tender.

Objectively a bulging of the posterior pharyngeal wall may sometimes be made out, or at least suspected, and on palpation fluctuation will be felt. The swelling is rarely central, and is usually limited to the oral region of the pharynx.

The chronic cases differ from the acute in the absence of continuous fever, though the temperature may be raised at night, and in the symptoms being less acute.

In adults difficulty and pain in deglutition are the chief

symptoms, and there is less difficulty in detecting the bulging abscess.

Diagnosis.—*Acute retro-pharyngeal abscess* in children is easily mistaken for croup, but the fixation of the head, the unilateral swelling of the glands, and the fact that deglutition as well as respiration is rendered difficult, should lead to a careful inspection of the throat.

The chronic form has to be differentiated from sarcoma, which grows rapidly, does not fluctuate, and but rarely causes fixation of the head.

Prognosis.—This should be guarded in children, because there is always the possibility of an underlying caries of the cervical vertebrae, and if symptoms of laryngitis are present there is risk of asphyxia or of secondary pulmonary complications; and in adults, because it is sometimes impossible to exclude the presence of phlegmonous pharyngitis. As a rule recovery is complete and rapid on evacuation of the pus.

Treatment consists in evacuating the pus as soon as fluctuation is detected, either through the mouth by aspiration or by the knife, or by an incision behind the sterno-mastoid. In one case I found the abscess pointing behind the left sterno-mastoid. When this was opened by a surgeon the retro-pharyngeal abscess became evacuated. When the abscess is opened by the mouth the patient's head should be very low, an assistant holding the head, pressing gently with the fingers on the post-maxillary region, while the operator guides the pharyngotome held in the right hand by the forefinger of the left hand. The incision should be made into the most dependent part of the abscess, and then the patient turned on his face to prevent the pus escaping into the larynx. A suitable antiseptic mouth wash should be used for some days.

The great danger lies in the occurrence of cedema of the glottis. Ice should be sucked if the patient is old enough, and hot applications made to the neck and submaxillary region. For the treatment of laryngeal complications, see *Cedema of the Larynx*.

CHRONIC PHARYNGITIS.

There is no pathological condition of the pharynx and fauces which is so difficult to define briefly as chronic pharyngitis, indeed the physical signs of the disease are very frequently

observed in those who do not complain of discomfort in the throat; we must therefore recognise that there is no constant relation between the objective signs and the symptoms, being guided in making a diagnosis of disease chiefly by the subjective symptoms.

For clinical purposes we distinguish three varieties :—

- 1.—*Simple chronic catarrhal pharyngitis.*
- 2.—*Chronic granular or hypertrophic pharyngitis.*
- 3.—*Atrophic pharyngitis or pharyngitis sicca.*

A fourth variety has been described as *exudative pharyngitis*, in which the lymphoid follicles are distended by a collection of cheesy secretion, but this is merely an accidental retention of the secretion and epithelium which has undergone fatty degeneration and which has not, as usual, been extruded.

Etiology.—The causes of chronic pharyngitis are very numerous and diverse. Thus it may be due to :—

1.—Recurrent acute attacks in adults. The characteristic objective features are frequently found in children, especially in those who are subject to hypertrophied tonsils and post-nasal adenoids and enlarged lymphoid nodules in the posterior pharyngeal wall. It is, however, unusual for subjective symptoms to arise in early life, but when the condition persists in adult life it may be the source of much discomfort.

2.—Nasal obstruction and consequent mouth breathing.

3.—A recent attack of measles, scarlet fever, or influenza, etc.

4.—The gouty or rheumatic diathesis.

5.—The nature of the occupation involving continual exposure to irritating vapours, tobacco dust, stone dust, mattress making, etc.

6.—The excessive use of tobacco, especially tobacco chewing, or free indulgence in alcoholic drinks; it is said also to result from the excessive use of irritating condiments.

7.—Dyspepsia, especially if associated with constipation and portal congestion, is a very common cause.

8.—General anæmia, more especially in chlorotic girls, is one of the most frequent and important causes of granular pharyngitis. In these cases other concomitant factors such as dyspepsia and constipation are generally present.

9.—The neurotic temperament is an important factor, and

accounts for the frequency with which "granular pharyngitis" has to be treated in females.

It is impossible to altogether separate the simple catarrhal and hypertrophic forms either as regards the etiology, symptoms, or physical signs; yet certain broad distinctions may be made. Thus, the first six causes enumerated generally result in a simple catarrhal pharyngitis, the result of more or less acute inflammatory changes. Chronic portal congestion, whether due to gastro-intestinal catarrh, or heart disease, likewise results in a chronic congestion of the pharyngeal mucous membrane. But there are many cases that must be regarded as toxic and due to a sluggish liver failing to arrest and destroy the toxic products of imperfect digestion, which have a specific effect on the pharynx and fauces like muscarin and belladonna. The redness and injection, the dryness of the throat and pain in deglutition, and the sense of roughness or presence of a foreign body in the pharynx, which are characteristic of belladonna or muscarin poisoning, are exactly imitated in dyspeptic pharyngitis, and though these symptoms often occur in a mild degree, their frequent repetition accounts for the permanent alterations in the structure of the mucous membrane. I would explain the pharyngitis due to lithiasis in a similar manner.

The etiology of *atrophic pharyngitis* is an open question. It may apparently result from long-standing hypertrophic pharyngitis, while in many cases it arises *ab initio*. It is very frequently associated with atrophic rhinitis, and is usually present in ozæna. We cannot too strongly insist on the state of the general bodily condition as an important factor in the causation of chronic pharyngitis.

Symptoms.—In simple catarrhal pharyngitis there is almost always a sense of weakness and discomfort in the fauces, with constant accumulation of tenacious mucus, which is hawked up with difficulty. The voice is readily tired, and is wanting in resonance owing to the relaxed muscles being hampered by the congested, thickened mucous membrane and accumulation of mucus. When complicated by nasal obstruction, or post-nasal adenoids, the alteration in the quality of the voice is especially marked, the higher notes in the singing voice being the first to suffer. The pharynx is remarkably irritable, gagging and

retching being very readily induced, and the uvula is very frequently relaxed and elongated or hypertrophied.

Pain in the throat, of a dragging character, on swallowing and especially at the commencement of a meal, is usually one of the chief complaints.

Patients generally suffer from a frequent irritable cough, partly the result of mucus accumulating in the larynx, but largely due to the hyperæsthetic condition of the mucous membrane exciting cough reflexly. There is a feeling of a foreign body, or of a hair in the throat which cannot be got rid of by hawking. A pharyngeal cough, often called a stomach cough, bears no relation to the amount of secretion to be expectorated.



FIG. 53.

Chronic pharyngitis, with elongated and hypertrophied uvula.

On examination, the mucous membrane of the pharynx is found to be diffusely congested, many enlarged veins are seen in the posterior pharyngeal wall, and some hypertrophied lymph follicles are always present. It cannot be too strongly urged that these appearances are often observed in patients who complain of no throat symptoms, while in others, who complain of all the subjective symptoms of chronic pharyngitis, the objective features may be comparatively insignificant.

The *velum palati* and uvula are relaxed and congested, and enlarged mucous glands are found dotted over their surface. The congested membrane is covered with tenacious mucus, and on examining the larynx, it is generally found to be similarly affected, especially the inter-arytenoid fold.

Occupying the posterior or lateral walls, lenticular, flat, ham-red granules are seen. These granules consist of proliferated adenoid tissue surrounding the orifices of the ducts of the mucous glands. In children and young adults such lymph nodules not infrequently exist without any symptoms, and are then of little or no importance.

In the mucous membrane between them, enlarged veins are often found. There is seldom any excessive accumulation of mucus; in fact, the mucous membrane is remarkably free from secretion, the patient complaining rather of dryness of the throat, and often there is remarkably little to be seen to account for the severity of the symptoms, and the weakness and alteration in the voice. Thus it is difficult to accept Michel's hypothesis that the toneless character of the voice, and consequent straining, are due to the uneven granules interfering with the resonator functions of the pharynx and naso-pharynx.

In some cases the bands of hypertrophic tissue are almost confined to the lateral walls of the pharynx behind the posterior pillars of the fauces, corresponding to the salpingo-pharyngeal folds, a variety known as *pharyngitis hypertrophica lateralis*.

Pharyngitis sicca may be regarded as the final stage of chronic granular pharyngitis, when the mucous membrane has undergone atrophy and thinning, both the mucous glands and lymph follicles having partaken in the atrophic process. A very similar condition occurs sometimes in diabetes.

In *chronic atrophic pharyngitis*, the thin, glazed, dry mucous membrane allows the colour of the subjacent constrictor muscles to be seen through it. The patient complains mostly of the persistent dryness of the fauces, as well as of the other symptoms mentioned above. When associated with ozæna, a few of the inspissated crusts of fœtid secretion may sometimes be seen behind the soft palate and in the larynx.

Treatment.—Chronic pharyngitis is almost invariably a secondary affection, and therefore when called upon to treat a case, we must always first determine whether it is due to nasal obstruction, or the presence of post-nasal adenoids, or to dyspepsia, heart disease, rheumatism, gout, anæmia, the excessive use of alcohol, and whether chronic constipation is associated with it. It is beyond the scope of this work to indicate the appropriate treatment of the general causes of chronic pharyn-

gitis, while insisting on the necessity for such treatment. The treatment of nasal obstruction from various causes is dealt with in another chapter.

The advisability of recommending a course of saline or aperient waters must depend on the nature of the case. Many chronic cases are wonderfully benefited by a period of residence at such Spas as Aix-les-Bains, Ems, Homburg, Mont Dore, La Bourboule, etc., or one of the health resorts on the northern African coast.

Local treatment is generally called for. The usual astringent lozenges and sprays are very inefficient and disappointing. If the mucus tends to collect in the pharynx and naso-pharynx, a solvent douche, composed of bicarbonate of soda or carbonate of potash (1 to 2 per cent.) with a few grains of boracic acid, may be used once or twice daily. Painting the parts with solutions of mineral astringents, such as sulphate of copper (10 to 20 grs.), nitrate of silver (20 to 50 grs.) or protargol (2 to 4 per cent.), sulphate of zinc, or the chloride (10 to 40 grs.), alum (20 to 60 grs.), or the anhydrous persulphate of iron (20 to 60 grs. to the ounce of water) is sometimes effectual. The application should be made daily, first with weak solutions, and less frequently as their strength is increased. An intelligent patient will soon get into the way of painting his own throat, although at first it is necessary for the physician to do it himself till a certain degree of tolerance is established.

A very useful gargle for general use in relaxed throat is a pinch of salt dissolved in a wineglassful of water.

Mandl's solution of iodine in glycerin is strongly advocated. It is used in strengths varying from 6 to 20 grains of iodine to glycerin 1 oz. Begin by using the weaker solution daily, and gradually increasing the strength with less frequent applications. It should be applied to the naso-pharynx and the whole of the pharynx; it causes a burning sensation lasting a few minutes.

Enlarged granular lymph follicles, if causing discomfort or other symptoms, should be destroyed by the galvano-caustic point. A somewhat flat-ended platinum cautery should be used, being placed against the centre of the nodule cold, the current turned on and withdrawn while still at a bright cherry-red heat, as soon as it begins to burn. Three or four granules may be

dealt with at a time. If there are any enlarged veins coursing over the pharyngeal wall, they should be divided in places by the galvano-cautery, so as to obliterate them. Of course such energetic measures require the previous application of 10 per cent. solution of cocaine to the parts to be operated on. This is especially necessary when, as in *pharyngitis hypertrophica lateralis*, the lateral walls of the pharynx require cauterising. Other methods of destroying the granular hypertrophy are advocated, such as touching the centre of the nodule with chromic acid fused on a silver probe, dividing them with a small knife, and inserting a small point of nitrate of silver for a moment, or even curetting. These methods are far less convenient than the cautery. Too much importance should not be attached to small nodules of lymphoid tissue; but enlarged veins, if left, may by maintaining the chronic vascular engorgement, render other therapeutic measures futile.

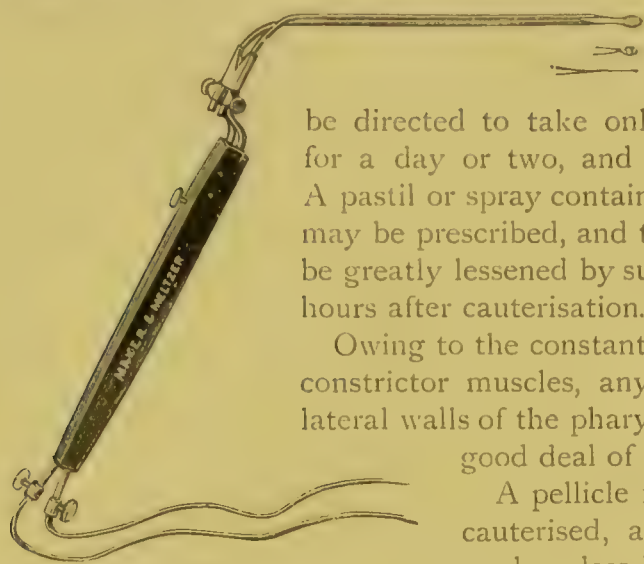


FIG. 54.
Galvano-cautery handle and lumen.

After using the galvano-cautery, the patient should be directed to take only bland, cold food for a day or two, and to avoid smoking. A pastil or spray containing a little cocaine may be prescribed, and the discomfort may be greatly lessened by sucking ice for a few hours after cauterisation.

Owing to the constant movements of the constrictor muscles, any operation on the lateral walls of the pharynx is followed by a good deal of pain on swallowing.

A pellicle forms on the point cauterised, and separates in a week or less, leaving a clean surface. The patient should there-

fore be directed to use some simple saline or cleansing spray or gargle for a few days, subsequent to the cauterising; and to come again in a week's time, when the treatment may be resumed if necessary. Generally several sittings are necessary before a "cure" is effected.

Curetting is sometimes advised for the rapid removal of hypertrophied follicles, while Schech and others have had good

results from excising thick and greatly hypertrophied bands, especially in cases where time was an object.

In clergymen, schoolmasters, and professional singers, any faulty method in producing the voice must be detected and corrected. In many cases a few lessons in the elements of elocution will do a great deal for a patient. The greatest difficulty will be encountered with professional singers who have been perfectly trained, whose speaking voice is apparently unaltered in resonance and strength, but who have lost the all essential strength and fulness in their highest notes only. This condition is often associated with a mild form of chronic granular pharyngitis; but while this may require local treatment in the pharynx, we must rely mainly on general measures, rest, and treatment directed to the larynx.

HÆMORRHAGE FROM THE PHARYNX AND LARYNX.

(SPURIOUS HÆMOPTYSIS.)

Hæmoptysis, a term which strictly implies spitting of blood, has by long custom been restricted in its application so as to refer only to blood derived from the bronchi or pulmonary tissue; when the source of bleeding is in the mouth, pharynx, or larynx, spitting of blood is termed "spurious hæmoptysis," or "bleeding from the throat."

Bleeding from the throat is important, not so much on account of the actual gravity of the condition, *per se*, as of the frequency with which so-called bleeding from the throat is really bleeding from the lung, and an indication of grave organic disease there.

Etiology.—The chief causes of *pharyngeal* hæmorrhage are: (1,) Various general affections and alterations in the condition of the blood which have a tendency to local hæmorrhages, viz., diabetes, albuminuria, syphilis, variola, typhoid fever, yellow fever, diphtheria, purpura, pernicious anæmia, leukæmia, hæmophilia, and mercurial stomatitis; (2,) Acute inflammations and ulcerations; (3,) Ulceration in syphilis, malignant disease, tuberculosis; (4,) Oozing of blood from spongy gums; (5,) Rupture of enlarged veins, *e.g.*, in gout and hepatic cirrhosis; (6,) So-called vicarious hæmorrhage in women at the menstrual period; (7,) Traumatism, foreign bodies, etc.

The chief causes of *laryngeal* hæmorrhages are much the same, but we may add excessive straining of the voice, especially

in singers, excessive muscular effort, and the bleeding after separation of crusts in laryngitis sicca.

(8,) Subglottic or tracheal hæmorrhage may arise from syphilitic, tuberculous or simple inflammatory disease, or from an aneurysm.

Finally, the blood may escape from the nose or rhino-pharynx into the mouth, due to any of the causes of epistaxis, post-nasal adenoids, fibromas, malignant growths, etc.

Symptoms and Diagnosis.—In slighter cases, such as oozing of blood from the gums, there may be only a slight taste of blood, which is spat out mixed with saliva. When the bleeding is more copious, or if even slight oozing occurs during sleep in the recumbent position, it may be hawked up mixed with frothy mucus and give the patient the impression it comes from the lung. In all such cases the mouth and upper respiratory tract should, if necessary, be thoroughly explored in order to discover the bleeding point.

But when hæmorrhage from the mouth is free, the blood may be poured out too rapidly for any such inspection; and then, if with the head held low, the blood runs out of the mouth without coughing or retching, the source of the blood is almost certainly from the mouth, pharynx, or nose.

The greatest difficulty arises with patients who, after a tickling in the throat, cough up blood mixed with frothy mucus, for on the one hand a little blood from the throat may be frothy and bright red, and on the other the blood in a copious hæmoptysis may be unmixed with air. The chief point of distinction between spurious and pulmonary hæmoptysis in these cases lies in the fact that in pulmonary hæmorrhage the patient continues to expectorate blood mixed with frothy mucus for a few hours, and that the sputum is often stained with altered hæmoglobin for some days.

The suspicion of tuberculous or other disease of the lungs should always be entertained, and evidence of its existence sought in the chest and in the general condition of the patient, for unless the bleeding point is actually observed in the mouth, nose, pharynx or larynx, it is never safe to exclude pulmonary disease from one examination, nor should the possibility of the bleeding being tracheal in origin be overlooked. (See also "Submucous laryngeal hæmorrhage.")

The **Treatment** is largely that of the particular dyscrasia which results in the hæmorrhage, but if the bleeding is copious and the source can be discovered, torsion of the bleeding vessel, or galvano-cauterisation, or the application of a paste composed of equal parts of tannic and gallic acid (by volume) mixed with a very small amount of water, may suffice to check it. It may be necessary to ligature the common carotid in hæmorrhage due to deep ulceration, or arising from inaccessible bleeding points.

CHAPTER IV.

DISEASES OF THE TONSILS AND UVULA.

ACUTE TONSILLITIS—CHRONIC HYPERTROPHY OF THE TONSILS—
TONSILLOLITHS—DISEASES OF THE LINGUAL TONSIL—
DISEASES OF THE UVULA.

Introductory Remarks.—At the junction of the upper parts of the alimentary and respiratory tracts which are developed from the epiblast (viz., the nose and mouth), with that beyond developed from the hypoblast, there is a ring of lymphoid tissue formed by the pharyngeal tonsil, the lymphoid tissue in the Eustachian lips and lateral pharyngeal wall, and continued below the soft palate by the faucial and lingual tonsils.

The diseases of these aggregations of lymphoid tissue in the throat are very similar, the differences in cause, symptoms, and treatment being mainly due to the different anatomical relations of these conglobate glands. The subject is therefore rendered simpler by grouping the tonsils together for the purposes of descriptions.

The tonsils are morphologically similar to the Peyer's patches in the intestinal tract, but their physiological rôle is not yet clearly understood. Stöhr has demonstrated that in their epithelial covering there are numerous gaps between the cells large enough to permit the passage of leucocytes, and an enormous transit of such cells undoubtedly occurs in the tonsils without destruction of the epithelial cells. It seems probable that these lymphoid structures, so constantly present and well-developed in early life, subserve some useful purpose as yet unknown, but there is little doubt that one of the chief functions is the arrest and destruction of pathogenic micro-organisms.

That absorption normally takes place in the surface epithelium and through the interfollicular lymph spaces in the tonsil, may be inferred from the observations of Goodall and Hendelsohn on the absorption of carmine and various powdered substances. Stöhr, Killian, and others considered that the tonsils also furnished leucocytes to the blood, but they are no longer credited with taking any part in blood formation. Various other theories as to the function of the tonsil have been advanced by different observers, but the only fact that appears to be clearly demonstrated is that the tonsils

exercise protective functions. Yet inasmuch as the tonsils all tend to atrophy in adult life while the need for such protection apparently still persists, this protective action is not the only purpose for their existence, and it may well be that they have other unrecognised functions to fulfil.

But if the tonsils are in some measure a protection against the invasion of micro-organisms, their protective power is limited, and once this limit be passed, they are a positive source of danger. The crypts and fissures of the tonsils may become "traps" for microbes, and the peculiar anatomical arrangement of their investing epithelium described by Stöhr opens the gates to their invasion into the tissues of the tonsil, whence through the lymphatic channels and vessels they may gain an entrance into the system; such systemic invasion by pathogenic micro-organisms frequently occurs. Recent researches by Von Babes on the pathology of pulmonary gangrene seem to prove that the gangrenous processes have for their point of departure not only the bronchi but "also the retro-pharyngeal glands and tonsils. Infection from the tonsils by the penetration of gangrenous products, which traverse the lymphatic vessels as far as the bronchial glands is the rule where the bronchi do not show traces of primary lesion. Such infection by the lymphatic vessels is undeniable, and is particularly acute and very diffuse."

Primary tuberculosis of the tonsils is less rare than is generally believed, and the failure of the faucial tonsils to arrest the development of the bacilli results in tuberculous cervical glands so commonly observed in weakly children. It is remarkable that the glandular affection seldom arises except during the earlier years of life before the tonsils usually atrophy.

The importance of this question of the tonsils being the channel of infection in a certain proportion of cases of tuberculosis is sufficiently obvious, and that such a danger exists appears well established by numerous researches.

It has been proved that tubercle bacilli may exist in apparently normal tonsils and are frequently present in those which have become hypertrophied (Störk, Büschke, Schlenker, Strassmann, etc.); Dieulafoy found that of 96 guinea pigs inoculated with pieces of enlarged tonsils and adenoids, no less than 15 developed general tuberculosis. Lermoyez also has recorded two cases of apparently simple post-nasal adenoids which histologically were shown to be tuberculous.

But, on the other hand, Jonathan Wright failed to recognise

evidences of latent tuberculosis in 121 faucial, pharyngeal, and lingual tonsils examined; and Hugh Walsham found no evidence of tubercle in a large number of adenoids and enlarged tonsils removed during life.

It thus appears that both healthy and simply hypertrophied tonsils in non-phthisical persons not infrequently contain tubercle bacilli which become entangled in the crypts and fissures, but that tubercular disease occurs in only a small percentage of cases, and mainly in those of a weakly constitution or of the strumous diathesis.

The cervical lymphatics become infected with tuberculosis either (*a*,) from the tonsil downwards, or else (*b*,) from the glands at the root of the lung upwards. In Krückmann's investigations of many fatal cases of pulmonary tuberculosis the evidence tended to show that the primary affection was in the lungs, and that the tonsils were infected by the sputum, but in children at any rate he considered the tonsillar infection was obviously the primary lesion. Sims Woodhead has also stated that he is "driven to the conclusion that this method of infection of the glands of the neck through the tonsils must be of comparatively frequent occurrence, especially in children living under insanitary conditions and subjected to various devitalising influences."

One other point is worthy of note here, viz.: that notwithstanding the enormous number of children who suffer from tuberculous cervical glands, it is *comparatively* rarely that phthisical patients show evidence of having had caseating or suppurating cervical glands. In a measure the tonsils and lymphatic glands are protective, especially when they exhibit marked local re-action.

There are numerous other pathogenic micro-organisms which have been demonstrated in apparently healthy throats, *e.g.*, streptococci, staphylococci, pneumococci,* leptothrix, and diphtheria bacilli. They tend to collect in the tonsillar crypts, and it seems indeed rare to find the streptococcus absent in any tonsil. Under conditions favourable to their growth, these micro-organisms cause disease which at first is more or less localised in the tonsil. Thus the researches of Zendziak and many others have shown that acute lacunar tonsillitis is due to direct infection by streptococci and staphylococci. From numerous observations it appears that the poison of rheumatic fever often gains entrance to the system by way of the tonsils, while in not a few cases endocarditis has so directly followed acute tonsillitis as to leave little doubt as to the former being the source of the cardiac infection.

*Bezancon and Griffon found pneumococci in the tonsils of every one of forty healthy persons.

We have seen already, that septic tonsillitis and acute phlegmon are often due to infection of the tonsils by the pyogenic cocci, but septic thrombo-phlebitis with pyæmic infarcts in the lung, etc., has also been known to arise from peritonsillar abscess. Again, in investigating the rôle of the tonsils in scarlatina, Dowson was led to the conclusion that tonsillar infection was the primary lesion, and that the tonsillar lesion and cervical bubo of scarlet fever were the analogues of the chancre and secondary bubo of syphilis. Kocher's observations show that even acute suppurative osteomyelitis may be due to pyogenic organisms gaining entrance through the tonsils.

To summarise :—

(*a*.) The lymphoid tissues in the pharynx, when healthy, exercise protective functions against the invasion of micro-organisms.

(*b*.) This protective function is limited, and particularly when the tonsils are diseased the tonsils are a positive source of systemic infection by various pathogenic micro-organisms.

(*c*.) That inasmuch as the tonsils normally atrophy in early adult life, while that tonsil which is least exposed to invasion by micro-organisms is most developed in young children (the pharyngeal tonsil), there is no relation between the development of the tonsils and the need for such protective function, which therefore is probably subsidiary and certainly not essential to health.

TONSILLITIS.

(Angina tonsillaris.)

Etiology.—Tonsillitis is essentially a disease of adolescence, but may occur at any time of life from early infancy upwards. It may be due to traumatism, as by injury by a spicule of bone in swallowing. Any condition tending to lower resistance to infection, such as ill-health, overwork, and especially chronic hypertrophy of the tonsils indirectly predisposes to attacks.

The affection is often due to cold and is most prevalent in damp or changeable weather. In my experience more cases occur in May than in any other month, and this month is proverbially treacherous for those who are susceptible to chilly weather. The influence of cold *per se* is a doubtful quantity, and though a chill diminishes the resistance of the affected region to infection, its effect is mainly due to living in close, ill-ventilated rooms in cold weather.

A good illustration of this is recorded by Busquet. On April 1st, a company of soldiers (about 100) went into barracks which had been recently disinfected very thoroughly. Within three weeks there were three cases of angina, and in the course of the next month eight more cases. Meanwhile seven other companies had arrived at the same camp on April 21st, but the men were lodged in tents. The nights were very cold and these men often could not sleep; they were obliged to walk about and stamp their feet; yet they remained almost entirely free from angina.

Acute tonsillitis is contagious, and several instances of tonsillitis spreading through a household have occurred in my experience.

The contagion usually takes place either by aerial convection or through milk; an infected milk supply sometimes causing an epidemic of tonsillitis. Not infrequently tonsillitis is due to septic infection, and the frequent occurrence of sore throats in members of a household should lead to the suspicion of bad drainage.

Pathology—The affection is directly due to the action of various micro-organisms, a series of my own cases yielding on culture, streptococcus longus and brevis, staphylococcus aureus and albus, diplococcus, tetracoccus, some short stout and some fusiform bacilli. Less frequently Friedlander's pneumococcus, or bacillus coli, is found.

But the Klebs-Loeffler bacillus is present in certain cases which throughout their course are "clinically" simple acute tonsillitis. The question sometimes arises as to whether these must be treated as acute tonsillitis or as diphtheria, but the only safe course is to regard them as diphtheria unless the diphtheria bacilli can be proved by inoculation of guinea-pigs to be non-virulent.

I am fully aware that some practitioners dissent from this view, but in support of my contention I may refer to the researches of Hermann Biggs, who, in a series of investigations on this very point, found that in nineteen cases in which diphtheria bacilli were present, but which were clinically lacunar tonsillitis or pharyngitis throughout their course, the bacilli were fully virulent when tested upon animals in no less than 17; while of 25 other cases (fully virulent diphtheria bacilli in all) commencing with the same clinical semblance of simple lacunar tonsillitis or pharyngitis, every one ultimately developed characteristic symptoms of diphtheria.

It is difficult to say what proportion of acute tonsillitis cases contain diphtheria bacilli, but I am convinced the proportion is small, and it seems probable that when paralysis follows tonsillitis it is usually diphtheritic and due to diphtheria bacilli in the tonsillar lesion.

The clinical importance of the supra-tonsillar fossa as a source of tonsillar disease has been demonstrated by Paterson, and I am able to confirm many of his statements from personal observation.

The origin and development of this fossa is described elsewhere (p. 38). The upper tonsillar crypts open into this space just above the tonsil, and when the aperture is large and free the extruded contents find their way into the mouth, but when the opening is relatively narrow, the fossa is frequently blocked by plugs of epithelium, leucocytes, mucus, lime salts, and bacteria, aggregated into cheesy masses, readily decomposing and becoming very offensive, so as to set up irritation or inflammation, while they may also lead to the formation of tonsilloliths.



FIG. 55.
The supra-tonsillar fossa much occluded by the plica tonsillaris.

It is a mistake, however, to suppose that suppurative processes in this region are always peritonsillar in origin. In a large proportion of cases the peritonsillar tissues are the primary seat of suppurative process, but in others the inflammation starts from within the tonsil, and extends in the direction of least resistance either towards the surface, or discharging into a crypt, or else towards the base of the tonsil with secondary formation of a peritonsillar abscess.

We may distinguish several clinical varieties :—

- (1,) *Superficial or lacunar tonsillitis*, with diffuse inflammation of the mucous membrane of the tonsil, and accumulation of fibrinous exudation and altered epithelium in the crypts which appears on the surface as discrete patches of yellowish exudation.
- (2,) *Parenchymatous tonsillitis*, in which the deeper tissues of the body are also inflamed, the amount of swelling being considerable.
- (3,) *Croupous tonsillitis*, when a false membrane forms on the tonsil.
- (4,) *Acute ulcerative tonsillitis*.
- (5,) *Phlegmonous or gangrenous tonsillitis*, in which suppuration and necrosis of the tonsil arise.
- (6,) *Peritonsillitis* in which the connective tissues above or in front of the tonsil are chiefly involved.

Suppurative tonsillitis when pus has formed) is especially prone to follow peritonsillitis, although parenchymatous tonsillitis is also liable to end in suppuration.

Symptoms. The general symptoms vary with the degree of inflammation present, generally commencing with soreness and stiffness in the throat for one day, with aching in the back and limbs, and a general feeling of malaise, followed by a rigor with rapid rise of temperature, which may soon reach 104° to 105° . The pulse is full and bounding, the tongue thickly coated, and the bowels constipated. The urine is of the usual febrile character, scanty, and not unfrequently albuminous. The spleen is often enlarged and sometimes the liver.

In not a few cases an erythematous rash appears on the surface of the body, either in patches or diffuse, resembling a mild scarlatinal rash, and this may be followed by fine powdery desquamation.

When the catarrhal inflammation extends to the rhino-pharynx and the Eustachian tubes, deafness and tinnitus will occur, or the deafness may be due to occlusion of the Eustachian orifices by the swelling of the pharyngeal tonsil.

The heart should always be examined and any symptoms of endocarditis noted, for valvular disease from rheumatic, or perhaps septic infection, may follow tonsillitis, while the arthritic symptoms and other manifestations of acute rheumatisms in greater or less degree are by no means rarely associated with tonsillitis. Catarrhal inflammation always extends more or less to neighbouring parts causing pharyngitis, or laryngitis, and sometimes spreading down the trachea. When it spreads upwards to the rhino-pharynx and involves the Eustachian tubes, deafness and tinnitus will occur. Either or both the pharyngeal and lingual tonsils are also involved in a considerable proportion of cases; Zendziak observed lingual tonsillitis in 12 out of 133 cases of faucial tonsillitis.

With the onset of inflammation and swelling in the tonsil considerable soreness and pain are felt, and in the severer cases, especially in peritonsillitis, the implication of the pharyngeal constrictor, styloglossus muscles, and other deep structures renders swallowing acutely painful, and accompanied by lancinating pains towards the ears. The submaxillary glands become swollen, salivary secretion is increased in amount, but owing to

the disinclination to swallow the saliva is often allowed to collect and drivel from the open mouth.

In acute lacunar and parenchymatous tonsillitis, the tonsil may at first be only slightly reddened and the appearance of the yellowish exudation in the form of round patches at the mouths of the crypts may be delayed for a day or two. Sometimes the crypts are gradually involved, commencing in the supra-tonsillar fossa and spreading downwards. The local symptoms are generally less severe, and the general disturbance more pronounced than in peritonsillitis, in which the local symptoms are especially marked and painful.

Suppuration very often follows peritonsillitis, though the other forms may end in this way. When a peritonsillar abscess has formed it causes a considerable smooth, red swelling in front of and above the tonsil, and it generally bursts above the tonsil or through the anterior surface of the palate, though sometimes the pus finds an exit through the posterior surface. The croupous variety is characterised by the formation of a pellicle on the surface of the tonsil, but the local and general symptoms are similar in other respects to the non-membranous forms.

ACUTE ULCERATIVE TONSILLITIS is characterised by the formation of one or more deep ulcers on the tonsils. When first seen, they are generally about the size of a threepenny-bit to a sixpence, the shape more or less round, with sharply defined, ragged, slightly raised edges, covered with grayish-white secretion readily separating, and which, on removal, leaves a red fungating surface. The ulcers are not surrounded by a hyperæmic zone; indeed, the tonsil is usually normal in size and aspect, or only slightly enlarged. Acute local symptoms, enlargement of the glands of the neck, and general febrile disturbance are usually absent.

The ulcers have been attributed to microbial infection; in one of my cases an agar-streak culture yielded streptococci only, and Wingrave has found streptococci, staphylococci and slender rods which stained faintly with methyl blue, similar to those observed by Lake; but Moure considers that they are the result of acute proliferation of the epithelial lining of the crypts, which, being retained and exerting pressure on the surrounding tonsillar tissues, causes local necrosis. They are seldom met with, except in weakly, anæmic patients.

Phlegmonous gangrenous tonsillitis is described in connection with other septic inflammations in the throat (p. 74).

Superficial and parenchymatous tonsillitis are generally bilateral, though one tonsil is more affected than the other, while peritonsillitis, the variety that most frequently terminates in suppuration, is often unilateral.

Diagnosis.—It is often a most difficult matter to differentiate between acute tonsillitis and diphtheria, especially in the absence of any definite membrane in the latter. In such a case the chief points in favour of diphtheria are low temperature, little pain, unilateral affection, and the presence of albumen in the urine. Submaxillary tumefaction is common to both diseases. In any doubtful case cultures should be made, especially when diphtheria is prevalent and the patient is exposed to contagion. If false membrane is present, it is generally yellowish, friable, and non-adherent when due to streptococci, and usually grayish-white, tough and adherent in diphtheria; but such distinctions are by no means absolute.

Acute ulcerative tonsillitis may be distinguished from chancre by the absence of stony hardness, hyperæmia and cervical bubo.

Prognosis. The *prognosis* of simple tonsillitis is nearly always favourable, but we must be on our guard lest, in the earlier stages, we mistake a more serious affection for tonsillitis, especially that rare but very fatal disease, acute infectious phlegmon. The prognosis is of course rendered more grave when symptoms of endocarditis arise in the course of acute tonsillitis. Nor should we be unmindful of the rare, though always, possible, occurrence of septic thrombo-phlebitis of the internal jugular vein with metastatic abscesses of the lung or pleural cavity, to which Ward has directed attention. Cases of death from suffocation in young children from excessively swollen tonsils are recorded, and tracheotomy has several times had to be performed to avoid asphyxia. Death has also occurred from rupture of a tonsillar abscess and escape of the pus into the larynx; and fatal perforation of the internal carotid artery, with ulceration of the internal jugular vein, has supervened in staphylococcal croupous tonsillitis.

All infective processes are prone to run a severe course in alcoholic subjects and in such patients; especially with indications of fatty heart and degenerated vessels, the prognosis must often be very guarded.

Treatment.—At the outset, in all cases the bowels should be freely moved by a saline aperient. If the temperature is elevated, the best plan is to give 10 to 20 grains of salicylate of soda every hour, or two hours, till the temperature is reduced, and the pain on swallowing is alleviated. Salicylic acid, salicin, or the soda salt, are universally advocated in cases where there is reason to suspect a rheumatic tendency, but in a great many patients where no rheumatic history, either family or personal, is obtainable, the effect of salicylic acid in abating the symptoms is most gratifying, and in my experience the soda salt is much to be preferred to salicylic acid or salicin. I usually combine three or four grains of quinine with the soda salt, and believe that while removing the mawkish taste of salicylate of soda, it favourably modifies and shortens the tonsillar infection.

R	Sodii salicylatis	grs. xij—xvj
	Quin. salicylatis	grs. iij—vj
	Mucil. tragac.	q. s.
	Aq. menth. pip. ad	ʒj

If the tongue is much coated, salicylate of bismuth may be added.

In young children aconite is an excellent remedy, drop doses of the tincture being given every hour till the temperature is normal. It is well to remember that chlorate of potash in large doses is a great depressant, and causes irritation of the kidneys.

Guaiacum, first suggested by Sir Thomas Watson, is advocated as a specific in tonsillitis by many authorities, and may be given either in powder or in the form of the ammoniated tincture, one teaspoonful in a half-glassful of milk, and is especially useful in cases of lacunar and parenchymatous tonsillitis.

Guaiacum lozenges may be prescribed in mild cases with advantage, and gargling with dilute solutions of chlorate or permanganate of potash, to which phenazonum may be added—10 or 15 grains to the fluid ounce. Sucking ice is usually comforting, and lessens the local inflammation.

If the pain and swelling are considerable, gargling may be out of the question, but a spray of cocaine (1 per cent.) and menthol (2 to 5 per cent.) in liquid paraffin tends to diminish pain. Pain on swallowing may be almost annulled by firm pressure immediately in front of the external meatus of the ear on either side—a valuable hint for which I am indebted to Mr. Mark Hovell.

I have found great benefit follow repeated painting of the tonsils with protargol solution (2 to 4 per cent.), especially if the supra-tonsillar fossa be syringed out once or twice daily with the solution mixed with warm water.

Incising the tonsils in one or two places sometimes gives relief; but the rapid diminution of pain and swelling by the free administration of salicylate of soda usually renders such a procedure unnecessary.

When suppuration has commenced, the inhalation of steam or gargling with warm water relieves the pain somewhat, and tends to make the pus "point." As soon as there is any indication of the spot where the pus may be found, a fairly deep vertical incision should be made behind the palate, not into the substance of the tonsil itself, but through the palate, in a direction from below, upwards and inwards, parallel with the free margin of the anterior pillar, as shown in *Fig. 56*.

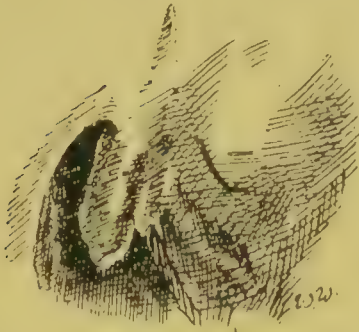


FIG. 56.
Suppurating peritonsillitis; to show
the proper line of incision.

The older method of incising the tonsil has been revived and extolled by Leland. He finds that by the use of the finger-tip, after the tonsil has been split by the sickle knife, the enlarged and distended crypt in which the suppurative process started can be frequently made out, and the sinus followed upward and outward through the tonsil into the peritonsillar cavity. The cavity is then freely torn open so as to secure free evacuation and drainage. He finds that the tonsillar inflammation not only rapidly subsides, but that, owing to complete drainage, it does not recur. The method is painful, and some general anæsthetic may be desirable.

The tonsils should not be removed while inflamed. To this two exceptions may be made: (*a*), when in children asphyxia is threatened; (*b*), in cases of chronic tonsillitis without hypertrophy, which cannot be removed during the periods of quiescence.

As the local symptoms subside a more tonic treatment is required, as the patient is often much weakened by the attack. It is well to advise the friends that tonsillitis, especially the

lacunar form, is contagious to some extent, and to avoid close contact, such as kissing; and when occurring in schools, the patient should be isolated from his companions.

CHRONIC ENLARGEMENT OF THE TONSIL.

Etiology and Pathology.—Chronic hypertrophy of the tonsils may be the result of frequently recurring acute attacks. It is often due to hereditary tendencies, and is frequently found in scrofulous children, and then generally associated with hypertrophy of the pharyngeal tonsil. Various exanthemata, particularly measles, are liable to cause enlargement of the tonsils, or to increase a pre-existing fulness. The condition may be met with at any age, but is rare after the twenty-fifth year, as the tonsils normally atrophy in early adult life.

We distinguish three varieties:—

(1.) *Chronic lacunar tonsillitis*, attended with accumulation of cheesy matter in the crypts, which gape when the yellow evil-smelling masses are extruded.

(2.) *Chronic parenchymatous hyperplasia*, chiefly found in scrofulous children.

(3.) *Chronic fibroid degeneration*, occurring generally in adults, and representing the advanced stage of the hyperplastic form.

Sometimes a pale yellowish swelling may be found, due to occlusion of the orifice of a crypt with retention of the cheesy exudation, one form of the so-called *chronic tonsillar abscess*. But a similar condition may be due to retention of secretions, etc., in a more or less closed supra-tonsillar fossa.

Symptoms.—The symptoms are local and general. With moderately large tonsils the chief complaint may be the frequent recurrence of acute or subacute attacks, which gradually undermine the health of the patient. When the hypertrophy is considerable, the voice is impaired, weak, with defective intonation and nasal twang, partly due to the mechanical interference with the movements of the soft palate, and partly to the chronic pharyngitis which generally co-exists. Respiration may also be much interfered with in children, with consequent defective aëration of the blood and deformity of the chest-walls, viz., pigeon-breast and infra-mammary depression of the ribs. Digestion too is often impaired, and the patient is generally anæmic and poorly developed. Dry reflex cough, darting

pains in the ears, and other reflex neuroses have been noted. Some difficulty in deglutition may often be noticed, but pain is uncommon, except when due to subacute attacks. When the faucial tonsils are much enlarged in children, post-nasal adenoids usually co-exist, and many of the symptoms generally associated with enlarged tonsils, *e.g.*, buccal respiration, snoring, deafness, facial and chest deformities, are really due to the enlargement of the pharyngeal tonsil.

The persistence of all these adverse symptoms, due to chronic tonsillar hypertrophy and the recurrence of acute attacks, keeps the sufferer in a perpetual state of general debility, and interferes with growth and development, both mental and bodily.

Diagnosis.—The tonsils often remain enlarged after an attack of acute tonsillitis, and it is necessary to be cautious in assuming that the hypertrophy will persist, till a week or two has elapsed. Apart from these cases, there is seldom any possible difficulty in making a diagnosis, the chief difficulty being to determine in the less marked cases whether the amount of hypertrophy is pathological. Two facts should be remembered: firstly, that a considerable hypertrophy may be concealed by the stretching of the anterior palatal pillar over the front of the tonsil, as shown in *Fig. 55*; and secondly, that the symptoms are not always proportional to the hypertrophy, many cases of moderate enlargement being associated with crypts which are distended with caseating plugs teeming with micro-organisms.

A unilateral hypertrophy may be due to malignant disease, either a sarcoma or carcinoma, and a unilateral enlargement of the tonsil commencing late in life should always lead to careful investigation on this point.

Prognosis.—As regards life, the prognosis is most favourable, but as regards health the outlook is very different. The tonsils very often begin to atrophy soon after puberty, and if that occurs, the patient may spontaneously “grow out of” the affection. But in a large proportion no such spontaneous cure occurs, and though the tonsils may undergo fibroid degeneration and shrink, they will probably interfere with the voice and comfort of the patient throughout the greater part of life. But even if such spontaneous atrophy does occur in early adolescence, that cannot bring back the years of childhood during which development has been retarded and the health undermined, apart

from all the suffering and interference with occupation and work, resulting from frequent intercurrent acute attacks and all their consequences. The necessity for operative interference for those cases which do not yield to milder treatment should hardly require further insistence, especially as the pernicious conditions can be readily and safely removed by a simple operation, and the prognosis be thereby rendered in all respects highly favourable. A healthy tonsil probably has some useful functions to fulfil, but a chronically diseased one never; indeed, it is not only a constant source of ill-health, but a positive source of danger.

Treatment.—The treatment of chronic follicular tonsillitis with cheesy accumulations and slight enlargement of the tonsil, is often very troublesome. A gargle of chlorate of potash and bicarbonate of soda will sometimes aid in the extrusion of the masses in the follicles. If there are only a few distended follicles the accumulations should be removed by pressure, or by means of a blunt probe, solid nitrate of silver or the galvano-cautery being applied to the empty follicle. Hoffman's plan of breaking down the walls of the follicles with a blunt probe is sometimes desirable. When the tonsil is enlarged it is simpler and more satisfactory to remove it.

For the hypertrophied and diseased tonsils there is in almost all cases only one method of treatment worthy of consideration—that is, removal.

There are various methods of removal: by cutting with a bistoury, scissors, or tonsillotome; by enucleation; by punch forceps (*morcellement*), or by the galvano-cautery snare or point.

I almost invariably use Fahnstock's guillotine or the galvano-caustic point. Mackenzie's tonsillotome is generally preferred,

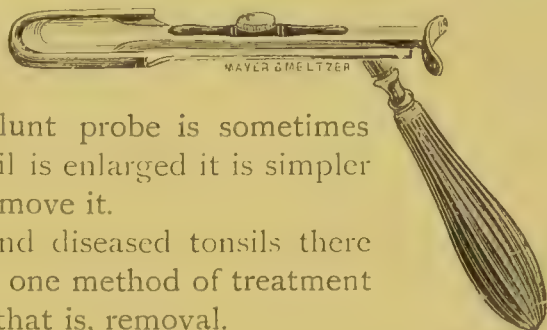


FIG. 57.
Mackenzie's
Tonsillotome.



FIG. 58. — Fahnstock's Tonsil Guillotine.

but an improvement on that instrument, and one I formerly used a great deal, is Reiner's, which combines in many respects the

advantages of the two first mentioned. But, in my experience, it is much easier to lift embedded tonsils out of the folds of the anterior pillar, and make certain a complete removal of all that is desirable with the old-fashioned guillotine. Whichever tonsillotome be used, it is well to gently press in the tonsil from the outside behind the angle of the lower jaw, so as to make the instrument engage it well. When the tonsils are very large this is unnecessary. It is important to direct the posterior part of the ring well behind the tonsil, otherwise an insufficient superficial slice only is removed, and a second attempt is necessary.

Galvano-cauterisation may be employed for reduction of enlarged tonsils; but it is a tedious and somewhat painful method, and should only be resorted to when a cutting operation is undesirable, as when there is reason to fear hæmorrhage in "bleeders," and in adults in whom the tonsils have become tough and fibrous, since there is then increased risk of secondary hæmorrhage.

A broad-pointed electrode should be used at a bright red heat. The process has to be repeated several times at intervals of three days to a week, till the tonsils are sufficiently reduced in size. The formation of cicatricial tissue completes the reduction.

In adults the application of a 10 per cent. solution of cocaine is the only anæsthetic required for tonsillotomy or galvano-puncture; but in young children the operation of tonsillotomy should always be done under nitrous oxide gas, bromide of ethyl, or chloroform, preferably the former.

The wire-loop and the galvano-caustic écraseur are still advocated for removal of the tonsils, mainly on account of the freedom from hæmorrhage ensured by these tedious methods. Morcellement consists in removing the tonsil piecemeal by special forceps. It is sometimes useful for small, irregular, ragged tonsils that cannot be guillotined. In using a bistoury, the tonsil should be drawn out and steadied with forceps, so as to facilitate removal by one sweep of the knife.

Enucleation is accomplished by separating the anterior surface of the tonsil from the anterior palatal arch, and then, with the tip of the forefinger applied to the upper border, tearing it out. It has been described as an easy proceeding, but I have never found it so, and now never attempt it.

Hæmorrhage is always pretty free after tonsillotomy, but

usually ceases spontaneously in a few moments. Dangerous hæmorrhage is rare when proper precautions are observed; but, on the other hand, it is liable to occur in any case. De Santi has reviewed the literature bearing on the question of hæmorrhage after tonsillotomy, and he finds that "*the Causes of Hæmorrhage after tonsillotomy are: (1,) abnormality in the distribution of the blood-vessels of the tonsils; (2,) hæmophilia; (3,) over-use of the voice; (4,) the too early eating of solids; (5,) the recorded cases of severe hæmorrhage have occurred very frequently after the use of the bistoury. Nearly all the cases of severe hæmorrhage from tonsillotomy have occurred in adults.*" But severe hæmorrhage also occurs in children and after the use of the tonsillotome. Moreover, the bleeding immediately after the operation may be only usual in severity and cease, and then, after a shorter or longer interval, burst out afresh, or continue to ooze for a long period without at any time amounting to free hæmorrhage. The practitioner should be on his guard against overlooking hæmorrhage when the blood is swallowed.

How Should we Treat the Hæmorrhage?—Most cases tend to cease spontaneously. The patient should be kept quiet and have small pieces of ice to suck. Should the bleeding persist, a mixture of one part gallic acid to three parts tannic acid dissolved in water may be sipped, or it may be applied in the form of a thick paste or bolus to the bleeding tonsil. Continuous pressure of the common carotid may be tried, or continuous pressure applied over the cut surface, especially when supported by counter pressure behind the angle of the jaw, and may be combined with the tannic and gallic acid paste; this almost always succeeds in stopping the bleeding. The pressure may be applied by means of a clamp with one extremity outside and the other inside. Another expedient is to pass a stout suture through the anterior and posterior pillars deeply, and then back through the posterior and anterior pillars, so as to encircle and constrict the stump of the tonsil. If these measures fail, the bleeding point should be sought for, and, if possible, seized and twisted with torsion forceps, or it may suffice to touch the bleeding point or surface with a cautery. Failing these and similar expedients, there remains, as a last resource, ligation of one of the carotid arteries, preferably the external carotid.

Cartilaginous and bony nodules.—Occasionally cartilaginous

nodules and bony trabeculae have been found in tonsils (Roth, Deichert and H. Walsham). Walsham considers that they are of foetal origin, that is, they are cartilaginous nodules derived from the second branchial arch. Kanthack, on the other hand, (remarks Walsham) dissents from this view, and considers that there is no embryonic inclusion, but merely a metaplasia of fibrous tissue into bone or cartilage. They appear to be of little importance, except when associated with enlarged tonsils and causing obstruction to tonsillotomy.

TONSILLOLITHS.

(Tonsillar Calculi, Calcareous Concretions.)

Etiology and Pathology. — These may occur either (1,) as small multiple accumulations of calcareous matter occupying the deeper recesses of the crypts, or (2,) in the form of large, generally single calculi.

Grüning has shown how they may be originated by the leptothrix buccalis in the tonsillar crypts, much in the same manner as tartar is deposited on the teeth; the nucleus thus formed accumulates around it mucus, pus and epithelium, which in time degenerate and become calcareous. Aitchison Robertson suggests that the larger calculi probably result from the retention of pus in the interior of the tonsil, the residual matter undergoing caseation and subsequent calcification. Others affirm that they are of gouty origin, and very rarely “chalk stones” of urate of soda have occurred. These, however, differ in composition from what are usually known as tonsilloliths, which consist chiefly of phosphate and carbonate of lime. Subjoined are two analyses of tonsillar calculi:—

LANGIER.					AITCHISON ROBERTSON.				
Water	-	-	-	25.0%	Organic matter	-	-	-	18.40%
Phosphate of lime	-	-	-	50.0%	Inorganic matter	-	-	-	81.60%
Carbonate of lime	-	-	-	12.5%	Phosphoric anhydride	-	-	-	50.00%
Mucus	-	-	-	12.5%	Calcium and magnesium	-	-	-	
				-	oxides	-	-	-	21.20%
				100.0%					

They usually vary in size from that of a pea to a filbert, but sometimes attain much larger dimensions. The largest yet recorded is one figured by Robertson, measuring $1\frac{3}{4}$ inches by $1\frac{1}{2}$ inches, weighing nearly one ounce.

Symptoms.—A moderate-sized calculus may cause no subjective symptoms, though generally giving the sensation of an enlarged tonsil, and often keeping up chronic inflammatory trouble and simulating chronic hypertrophy. They are liable to cause suppuration, and, with the rupture of the abscess, the calculus may suddenly escape and become lodged in the larynx, causing fatal asphyxia. Such an accident nearly asphyxiated Robertson's patient.

Diagnosis.—When small and embedded in the tonsil, it may be difficult to detect the existence of these concretions; but when less deeply situated careful palpation and the use of a probe generally suffices to reveal their presence. A unilateral enlargement of the tonsil may be due to chronic hypertrophy, malignant disease, or a tonsillolith.

Treatment.—When detected the concretions should always be removed by forceps, after incision of the tonsil, if necessary.

DISEASES OF THE LINGUAL TONSIL.

The lingual tonsil is developed later than the faucial and pharyngeal tonsils, and in early childhood is often small and ill-developed; thus its diseased conditions are more prone to appear in adult life.

The lingual tonsil is subject to the same diseases as the faucial tonsils, and may therefore be the seat of *acute lacunar* or *parenchymatous tonsillitis*, which may go on to suppuration. It very often participates in a *pharyngo-mycosis*. The treatment is the same as for these affections in the faucial tonsils.

Chronic enlargement of the lingual tonsil is sometimes developed, and the hypertrophied mass may overlap or impinge against the upper surface of the epiglottis, causing, in some of the cases, troublesome cough and a sense of persistent discomfort in the throat, globus, tenesmus, vocal impairment, etc.

It is necessary here to refer to a condition to which undue importance is sometimes attached, viz., the superficial veins in the dorsum of the tongue. It has been stated that a varicose

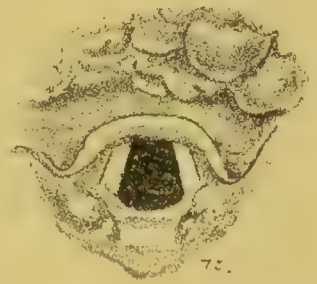


FIG. 59.
Hypertrophied adenoid tissue in the glosso-epiglottic fossæ. (Laryngoscopic appearance)

enlargement of these veins, the so-called *lingual varix*, is the cause of numerous symptoms and much discomfort; but inasmuch as this condition of the veins is very frequently present in persons who complain of no throat symptoms whatever, and is so frequently present in people over fifty as to be almost a normal condition, it is difficult to see how these veins can really be the source of all the troubles which have been attributed to them.

When considerable hypertrophy of the tonsil is associated with definite symptoms in the throat which call for relief, its removal should be effected either by repeated applications of iodine (Lugol's solution), by the galvano-cautery, or by means of a lingual tonsillotome.

A *Lingual Accessory Thyroid gland* is occasionally developed forming a round, smooth, firm, readily-bleeding tumour at the base of the tongue in front of the epiglottis, and corresponding to the foramen cæcum, covered with unchanged mucous membrane. These accessory thyroid glands are developed from the foetal thyroglossal duct (see p. 36). Cases are recorded by Melbraith, Reintjes, Watson and others. The symptoms are very similar to those of chronic hypertrophy of the lingual tonsil, and the two conditions may easily be mistaken for one another. The recognition of this accessory thyroid gland derives importance from its sometimes performing the functions of an otherwise absent thyroid, and in one case its removal was followed by well-marked myxœdema within seven months (Seldowitsch). On the other hand, a lingual accessory thyroid has been found in a cretin (de Boncourt).

If large enough to cause definite symptoms, treatment should first consist in the administration of thyroid gland internally. Should this fail to relieve, and the normal gland is not absent, extirpation by the galvano-cautery loop, enucleation through the mouth, or removal through an external incision may be necessary.

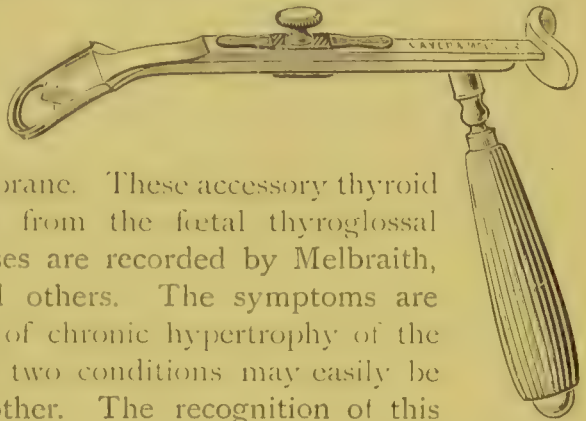


FIG. 60.
Brady's lingual
Tonsillotome.

DISEASES OF THE UVULA.

The uvula being practically a portion of the soft palate is generally more or less implicated in affections of this region, yet its diseases present certain special features requiring notice.

Congenital defects are not uncommon, the uvula being absent, or bifid in part or the whole of its length, or twisted and hanging to one side. These defects are of no practical importance, except in so far as they may be mistaken for evidence of disease which does not exist, *e.g.*, syphilis, paralysis, etc.

Inflammatory affections may cause considerable swelling and elongation of the uvula. They are generally associated with faucial inflammation, but traumatic or septic uvulitis may occur alone.

Elongated Uvula. — Pathology.—The uvula is almost invariably found to be more or less relaxed in chronic pharyngitis; and whereas the elongation in some cases gives rise to no symptoms, it generally increases the unhealthy condition apart from any of the exciting causes which initiated the mischief, sometimes producing alarming symptoms and requiring special treatment. It is necessary, however, to enter a protest against the habit of some practitioners of snipping off the uvula in all and sundry cases.

In some cases the uvula and soft palate are merely relaxed, otherwise remaining normal in appearance, there being no congestion or hypertrophy; in others there is hypertrophy and chronic congestion of the soft palate and fauces, often resulting in degeneration of the glandular structures of the rhinopharyngeal mucous membrane, and associated with severe constitutional disturbance.

Symptoms. In the simpler cases, where there is merely relaxation of the uvula and soft palate without hypertrophy or congestion, the symptoms are mainly impairment of the quality and strength of the voice, and are chiefly observed in professional singers.

In a marked case the patient usually complains of continual hawking, with a feeling of some foreign body in the throat that cannot be coughed up, often likened to a hair or fish-bone in the throat. The cough may be very severe, particularly at night on lying down. The constant titillation at the back of the tongue

not infrequently results in vomiting, this especially in the mornings or after meals. When the uvula is so elongated as to reach the larynx, laryngeal spasms occur in consequence.

Diagnosis. In doubtful cases the patient should be directed to open his mouth, and breathe quietly. At first the uvula will be

partially retracted into the soft palate, but if elongated it soon drops, and the tip rests on the back of the tongue. On striking a high note the normal uvula is almost completely drawn up into the soft palate which is raised (*vide Fig. 61*), but the relaxed uvula is shortened in wrinkles of redundant mucous membrane (*vide Fig. 63*). The redundant trans-

lucent mucous membrane is generally obvious along the free edges of the velum and at the tip of the uvula.

An example of the elongated and hypertrophied uvula, such

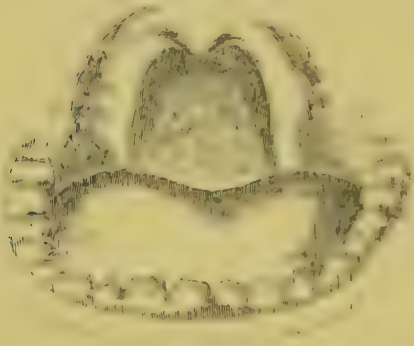


FIG. 61.
Retraction of the normal uvula on striking a high note.



FIG. 62.
Elongated uvula.

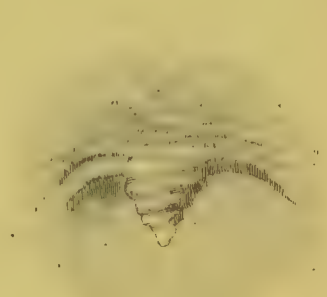


FIG. 63.
The same, on striking a high note, showing the abnormal wrinkling of the redundant mucous membrane.

as is generally associated with chronic pharyngeal catarrh, is shown in *Fig. 53*, from another patient.

Treatment. In treating cases of relaxed uvula it is well to give local astringent applications a fair trial, especially in the milder cases.

When local applications have failed the uvula ought certainly to be partially removed. The great amount of benefit that may result from such a simple procedure as removal of the uvula was well illustrated by a case under my care at the Bristol Royal Infirmary. When admitted this case appeared to be far gone in consumption, and, in fact, had been treated for tuberculosis of the lung by his doctor. He was very feeble and emaciated, and *râles* were detected over the whole of both lungs. His uvula was partially ablated, as symptoms pointed to elongated uvula, and the subsequent improvement and final recovery were rapid. He gained 3 lbs. in weight in a fortnight. It is not surprising that the loss of sleep and frequent vomiting should result in great emaciation and weakness. Such wasting associated with cough and expectoration of mucus streaked with blood from the pharynx, might lead one to suspect that the patient is suffering from tubercular disease of the lungs, especially in those who also complain of localised pains in the chest—pains which are purely reflex in origin. The amount of blood that may be lost in this way is very considerable.

The parts having been cocaineised, the tip of the uvula should be seized with forceps and gently drawn forward. The redundant portion is then removed by one cut with a pair of curved, blunt pointed scissors. By operating in this way the cut surface is situated posteriorly, and is not irritated by food on deglutition.

I have never seen any but good results from the procedure. In one patient, however, the son of a medical friend, there was severe secondary hæmorrhage three days after the operation; the possibility of such an occurrence should be borne in mind, and a warning given to the patient or friends. The “*gargarisma acidi tannici et gallici*” (see *Formulæ*) is useful in case of hæmorrhage.

The food should be soft, bland, and cold, for a few days after the operation, and a mild morphine and cocaine lozenge should be sucked at intervals, especially before meals.

Paralysis, new growths, and various infective diseases such as syphilis, tubercle, etc., may involve the uvula, but these are described in connection with similar conditions of the pharynx.

CHAPTER V.

MEMBRANOUS ANGINA AND DIPHTHERIA.

MEMBRANOUS ANGINA DIPHTHERIA OF THE FAUCES, LARYNX AND NOSE.

MEMBRANOUS OR DIPHTHEROID ANGINA.

(PSEUDO-DIPHTHERIA.)

Etiology and Pathology. The false membrane of croup, and that resulting from scalds and caustics, is composed of coagulated fibrin, degenerating leucocytes, proliferating connective-tissue and epithelial cells, and, in most cases, non-pathogenic micro-organisms are present. In other cases the formation of the false membrane is due to a variety of pathogenic organisms, e.g., streptococci, staphylococci, Vincent's spirillum, bacterium coli, etc.

Streptococcal membranes are occasionally formed in scarlatina, small-pox, and typhoid fever. A pure streptococcus pseudo-membrane may generally be distinguished from a true diphtheria from its being yellowish in colour, and friable, instead of greyish-white and tough; nevertheless, it may resemble a typical diphtheritic membrane in all respects. Acute streptococcus infection usually takes the form of acute follicular tonsillitis, with swelling of the glands behind the angle of the jaw, which may suppurate; symptoms of septicæmia, purulent pleurisy or pericarditis, etc., may supervene. There is generally considerable pain in the throat, the temperature ranging about 102°0 to 104°0; the spleen may be enlarged, and the urine albuminous. A scarlatiniiform erythema, diffused or in patches, is often present, and may be followed by powdery desquamation.

Regarding the appearance of false membrane in true scarlatina, Klein, as the result of his recent researches, confirming the conclusions of Babes, Holzinger, and others, lays down the following *general* rule: "Diphtheritic symptoms appearing as a sequel to scarlet fever, *i.e.*, in one to two or three weeks, are true diphtheria; but the like symptoms occurring as a complication, *i.e.*, in the course of the fever, are not, and though the

streptococcus of scarlet fever may be present, we shall not find 'Loeffler's bacillus.'"

A pure staphylococcus infection may apparently exactly simulate true diphtheria in its clinical aspects, a case in point being reported by Glover, in which the grey pseudo-membrane extended to the larynx and down into the bronchi, ending fatally; the same remarks apply to the spirillum infections.

The spirillum form of diphtheroid angina described by Vincent, generally affects one tonsil only, and the neighbouring fauces. At the onset the tonsil is covered with a white or grey, thin and soft pseudo-membrane, easily detached but leaving an eroded bleeding surface. About the third or fourth day the membrane is found to be thick, soft, and almost caseous superficially, the breath is fœtid, and the adjoining mucosa œdematous and red. As a rule the submaxillary lymphatic glands are a little enlarged, but do not suppurate, and there is usually slight febrile disturbance. The membrane generally clears up about the eighth or tenth day. In children the disease is very fatal. Both a bacillus and a spirillum are found in these cases. The bacillus is fusiform in shape, 6-12 μ in length, and may be straight or curved. It stains readily with the aniline dyes, and involution forms show non-staining vacuoles; it does not stain with Gram's method. It is best demonstrated by preparing a film by rubbing a piece of the membrane on a cover-slip; the bacilli are then seen to be present in great numbers, accompanied by a spirillum. These organisms have not as yet been cultivated on artificial media, nor have they been shown to be pathogenic to animals. The fusiform bacillus is a normal inhabitant of the buccal cavity.

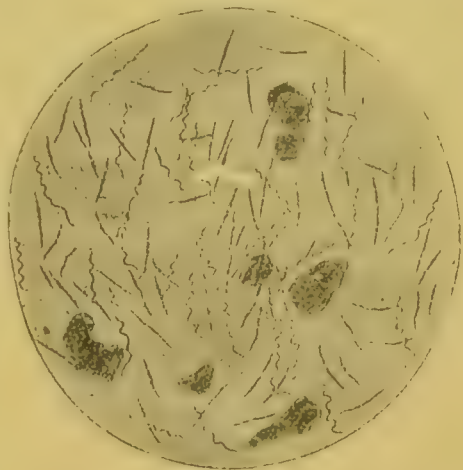


FIG. 64

Spirillum and fusiform bacilli. VINCENT.
(From the "Ann. de l'Inst. Pasteur.")

Similar cases have been recorded by Bosquier (with presence of micrococcus tetragonus, streptococci, some spirilla, and Vincent's fusiform bacillus), and Schneider; in both there was

ulcerative tonsillitis—in Schneider's case resembling syphilitic ulceration, but rapidly clearing on local treatment. Craig found large spirilla 20 to 35 μ long, resembling spirilla of relapsing fever, in a case of subacute tonsillitis, with thick, dirty-white membrane.

The most successful treatment appears to be painting with iodine solution and using boric acid gargles.

The *Bacterium coli* has also been known to cause acute and chronic tonsillitis, with a dull, whitish, soft exudate covering the tonsillar crypts. The intervening surface was red and free, but the membranous coating was slightly adherent, though no bleeding occurred on its removal. The symptoms began with a short period of acute tonsillitis, followed by a prolonged chronic course. The local subjective symptoms were comparatively slight, but disorders of the alimentary tract and deterioration of the general health were notable features. The exudate yielded pure cultures of the *bacterium coli*. [For "Non-Diphtheritic Croup" and "Fibrinous Rhinitis," see chapters viii and xii.]

DIPHTHERIA.

Definition. Diphtheria is a specific infectious disease, due wholly or in part to the Klebs-Loeffler bacillus, and primarily a local infection, generally commencing in the fauces, more rarely in the larynx, nose, or genital mucous membrane, or on the surface of a wound. Being essentially a local infection the initial symptoms are local, and accordingly cases may be classified as pharyngeal, laryngeal, nasal, or wound diphtheria, etc.

The difference between croupous and diphtheritic inflammation is now known to be merely one of degree, and the same infective micro-organisms may give rise to either. Moreover, several varieties of micro-organisms, and various chemical irritants, are known to cause inflammation with false membrane and superficial necrosis, anatomically identical with Virchow's definition of diphtheritic inflammation, while on the other hand the specific disease associated with the Klebs-Loeffler bacillus may be unattended with the presence of any membranous exudation at all.

It would be convenient, and prevent much confusion, if the term diphtheria could be employed in a clinical sense to include all affections characterised by the presence of false membranes, their pathological distinction being indicated by such terms as

staphylococcal, streptococcal, mixed Loefflerial, pure Loefflerial, scarlatinal, traumatic, etc. But the word diphtheria has become definitely associated with the disease due to the Klebs-Loeffler bacillus, and the term must be reserved for all its clinical variations, whether the resulting local inflammation be attended with the formation of false membrane or not. Every inflammation due, partly or wholly, to the Klebs-Loeffler bacillus is diphtheria, though the mere presence of the specific bacillus in the throat does not constitute diphtheria, for it sometimes occurs in healthy throats, such a condition being exactly analogous to the presence of leptothrix spores or mycelium, streptococci, staphylococci, or even tubercle bacilli in a normal throat.

Etiology. Children between the ages of three and fifteen are especially liable to contract diphtheria when exposed to infection, and it is in these first fifteen years of life that the great majority of deaths occur.

The influence of season on the number of fatal cases from diphtheria is fairly constant, but the influence of school attendance must be held to partly account for the increase in the number of attacks in the spring and autumn.

Coincident with sanitary progress is the development of education amongst the masses, and there is ample reason to attribute the increased incidence of diphtheria in a large measure to the more general and regular attendance at day and Sunday schools.

The influence of sewer gas and insanitary environment is a moot question, but facts do not support the still very prevalent opinion that bad sanitation is directly a common cause of diphtheria, although it may have some indirect influence. Improved sanitation, which has characterised the last quarter of the nineteenth century, has been followed by a remarkable diminution of diseases that are known to depend on defective sanitation, viz., typhoid and typhus fevers; but during the same period there has been a progressive increase in the incidence of diphtheria, especially in towns where the progress in sanitation has been most marked.

Nevertheless the influence of sewer-gas may operate by causing sore throats, and thus rendering those exposed more susceptible to the infection by the diphtheria bacillus.

Conveyance of Infection.—Infection generally occurs by aerial convection, either directly from expectorated particles from an

infected individual, or from fomites, books, or playthings that have been exposed to contamination, or by direct inoculation by kissing, etc. A number of epidemics have been definitely traced to milk. But there is no doubt that infection may be carried by apparently healthy persons, numerous instances of this having been observed and recorded; for example, nurses and others who have been exposed to infection, or convalescents after attacks of diphtheria may carry the specific bacillus in their throats and, without showing any symptoms of the disease themselves, may communicate diphtheria in a virulent form to others. Another often obscure source of infection is latent diphtheritic fibrinous rhinitis (see *page* 139), and instances have occurred where a person in apparent health, but in whom fibrinous rhinitis has been found, have afforded the only possible source of a virulent attack of faucial diphtheria in those coming into close relations with them.

Cats have been shown to suffer from diphtheria, and may then infect human beings. The diphtheria bacillus has been found by Klein in the patches of broncho-pneumonia in cats suffering from diphtheria; false membrane does not appear to be formed in the cat, the disease mainly affecting the lung, and causing also kidney affections and ophthalmia. The only other domestic animal known to become affected with diphtheria is the cow, in which the disease is known as "chapped teats." The false diphtheria-like membranes sometimes found in pigeons and turkeys are due to organisms, but the Klebs-Loeffler bacillus has not been identified in connection with them.

Prophylaxis.—Inasmuch as any inflammatory or diseased condition of the mucous membrane of the upper respiratory tract renders an individual more susceptible of infection by the diphtheria bacillus, it is desirable to remove susceptible individuals from damp or insanitary surroundings, and combat any existing disease in the nose and throat by appropriate measures. Especially should chronically-enlarged or hypertrophied tonsils be attended to.

During an epidemic of diphtheria all milk should be sterilised by boiling, or by heating to 140 F. for five minutes; the clothing of those attacked should be carefully disinfected; the floors, walls and furniture of their rooms disinfected by formalin spraying.

The importance of isolating patients who have recently had diphtheria, at any rate so long as diphtheria bacilli virulent to guinea-pigs can be obtained from the throat or nose, is obvious, for no one can say that the bacilli, even if shown to be of slight virulence at the time, will not cause a severe or fatal attack in others who may be inoculated from them. Particularly is this rule important with regard to schools, for I cannot understand why it should not be considered necessary to be as careful in regard to diphtheria as in scarlatina. No practitioner would take the responsibility of permitting the mildest case of scarlatina or an ambulatory typhoid to mix with others in a school until absolutely free from infection, and so long as Klebs-Loeffler bacilli persist in the throat of any child it ought to be regarded as potentially infectious and separated accordingly, at any rate until our means of determining the infective power of apparently non-virulent bacilli morphologically indistinguishable from the virulent Klebs-Loeffler bacillus is placed on a more satisfactory footing. If several cases of diphtheria break out in a school, the question of closing the school must be carefully considered. If an elementary school it should undoubtedly be closed, but in the case of elder children, swabs should be taken from each individual, and if any are shown to have the bacillus they should be promptly isolated. Again, after a school has been closed on account of an outbreak no scholar should be allowed to return without a bacteriological examination of the throat, whether there is a previous history of diphtheria or not. Nor should any scholar be allowed to return to school from a house where diphtheria has occurred until at least twelve days have elapsed after all those at home have become convalescent, nor until he has himself been subjected to a bacteriological examination.

It must, however, be admitted that such ideal conditions cannot always be maintained, in view of results obtained by Goadby from the simple bacteriological examination (without inoculation) of one hundred healthy children in a school in which no diphtheria had occurred for two years, for in forty-two of these children with unhealthy throats, fourteen showed the *b. diphtheriæ*; and in the fifty-eight with healthy throats, four showed the bacillus. Such results in a London school, which, though itself free from diphtheria, was in the neighbourhood of

other schools where diphtheria was rife, while explaining how diphtheria epidemics break out, show also how useless it would be to adhere too stringently to the rules laid down for schools where diphtheria bacilli are not prevalent in the apparently healthy. The numerous bacteriological cultures I have made from boys with sore-throats at Clifton College, have never once yielded a diphtheria bacillus, and in such normal schools one should avoid accepting the presence of diphtheria bacilli as compatible with school attendance.

Post-scarlatinal diphtheria is especially prone to occur in hospitals for infectious diseases. Garratt and Washbourn, at the London Fever Hospital, succeeded in largely reducing the percentage of post-scarlatinal diphtherias by separating those in whom diphtheria bacilli were found.

Antitoxic serum injections have been given with considerable success for the purpose of producing immunity. Klein and others have been able to render guinea-pigs immune by antitoxic serum injections, but complete immunity in guinea pigs and in human beings appears not to extend beyond two or three weeks. The value of protective inoculations is insufficiently recognised as yet, but it seems to have been strikingly exemplified during various epidemics.

Bacteriology. The diphtheria bacillus is extremely polymorphic, and presents many variations in size and shape—now long and curved, now short and straight, one time clubbed at the end, another swollen at the centre with pointed ends, and while nearly always presenting characteristics sufficiently marked to enable an expert to differentiate it from other micro-organisms, its identification may sometimes cause considerable difficulty, and the difficulties are increased by the close resemblance of certain other non-diphtherial bacilli. The bacilli contain no spores, have no flagella, and are immobile. They always arrange themselves in more or less parallel groups or in clusters at various angles, and they never form chains. Two varieties may be described, the long and the short pathogenic: (1.) The long form, sometimes called “typical” diphtheria bacilli, are straight, slightly curved or *J*-shaped segmented rods, generally clubbed at one or both ends, of varying length and thickness, and often grouped in parallel bundles of two or more. The ends, if not clubbed, are rounded. When stained they have a

granular or segmented appearance from taking up the dye more deeply in parts, and when only one or two segments of a bacillus are stained, the "granules" correspond with the ends of the bacillus—"polar staining"; (2,) The short form generally occurs as short, slightly curved, or straight rods, in parallel groups of two or more, slightly swollen and deeply stained at one end, the other end tapering; or swollen in the middle, which is deeply stained, and pointed at the ends. Some of the bacilli with deeply-stained centre are diplo-bacilli—two bacilli still united end to end. Others are somewhat pear-shaped, and others form the "sheath bacilli" of Peters. The division of these bacilli into two groups is more or less arbitrary, as transitional forms will often be found in a culture which mainly yields the one or other of these groups. Nor can any reliance be placed on the view that the short form is less virulent than the long, though the pear-shaped variety is more usually an involution form. Kanthack stated "that after an extensive examination he can say confidently that it is futile to base a prognosis on the type of organism present." Some of the worst cases he has seen were associated with the short variety exclusively.

From experimental investigations on the toxin formation during the growth of the bacillus diphtheriæ, Kossel found that the maximum toxicity of a culture is reached in five days.

Bacilli resembling the Klebs-Loeffler Bacillus (so-called pseudo-diphtheria bacilli).—Certain bacilli which closely resemble the diphtheria bacillus are found not only in association with the Klebs-Loeffler bacillus, but in the normal secretions of the mouth, and on the conjunctiva in xerosis conjunctivæ or even in health.

By some the bacillus of xerosis is considered identical with Von Hoffmann's bacillus, by others it is believed to be a distinct micro-organism. Kober states that when cultures are examined within six hours, only an occasional xerosis b. is "plump," the majority are thin and easily distinguished from the Klebs-Loeffler bacillus.

Hoffmann's Bacillus has been the subject of much discussion. In a twenty-four hours' culture on agar or serum it appears as a short pyramidal bacillus, 1.5μ long, arranged in pairs, set end to end in parallel groups of two or more, that is to say, it is a diplo-bacillus. Its protoplasm stains more deeply at the apposed ends. It occurs in many forms of sore throat, and sometimes in association with the long or short forms of the true Klebs-Loeffler bacillus, but it is non-pathogenic to guinea-pigs, or, at most, causes a local inflammation at the site of injection.

Bacilli morphologically indistinguishable from the Klebs-Loeffler bacillus, but non-pathogenic to guinea-pigs, are regarded by some as a distinct form. Roux and the French school believe that the pseudo-diphtheritic bacillus is simply an attenuated form of the Klebs-Loeffler bacillus, which under proper conditions may regain its virulence. Loeffler, Escherich and the

German and English schools generally have regarded this bacillus as a distinct microbe. But Trumpp, and Hewlett and Knight, state that they were sometimes able to convert the non-virulent pseudo-diphtheria bacillus into a virulent Klebs-Loeffler bacillus; and it has apparently been possible by careful treatment to convert a typical Klebs-Loeffler bacillus into typical non-virulent pseudo-diphtheria bacillus, and more recently Salter, working with goldfinches, which are very susceptible, has apparently converted non-pathogenic bacilli, typically of the Hoffmann class, into typical virulent Klebs-Loeffler bacilli. It, therefore, seems hardly safe to assume that non-virulent bacilli, morphologically identical with the Klebs-Loeffler bacillus, is not *potentially* a true bacillus diphtheriae, but inasmuch as bacilli non-virulent to guinea-pigs, but morphologically resembling the bacillus diphtheriae, have frequently been isolated from noma, cancrum oris, impetigo, various chronic ulcerations, in numerous healthy throats, and by Cantley in ordinary acute febrile nasal catarrh (bacillus coryzae segmentosus), it is hardly practicable to regard the presence of such non-virulent bacilli as a sufficient reason for absolute isolation.

Furthermore, Von Babes has found bacilli morphologically similar to Klebs-Loeffler's bacillus in gangrenous tonsillitis and pulmonary gangrene: this diphtheroid bacillus was obtained (amongst other organisms) in ten out of twenty-four cases of pulmonary gangrene, and Babes attributes the gangrenous process in some measure to this bacillus.

Detection of the Bacillus. *Direct Examination.* A diagnosis can sometimes be made by the direct examination of a cover-glass film, and, pending the more reliable result obtainable from the examination of artificial cultures, is worthy of trial. The staining of the film should be made very shortly after taking the secretion or membrane from the throat, as saprophytic organisms rapidly multiply, and crowd out the diphtheria bacilli. The diphtheria bacilli may be stained by methylene-blue or carbol-fuchsin, and they are not decolourised by Gram's method. Artificial cultures may be made on serum, agar, serum agar, glycerin agar, gelatin, glucose, bouillon, litmus milk, or litmus whey (see *Plate XII*).

Kanthaek and Stephens recommend an easy method of preparing serum-agar in less than an hour. To every 100 c.cm. of serous exudation—ascitic, pleuritic, or hydrocele fluid—2 per cent. of a 10 per cent. solution of caustic potash must be added. To this they add 1·5 to 2 per cent. of agar-agar, previously soaked in acidulated water, and then boil the mixture in a Koch's steamer till the agar-agar is well dissolved. It is then filtered through a hot-water funnel, and to the filtrate 5 per cent. of glycerin and 1 per cent. of grape-sugar should be added. It may then be poured into test-tubes, and after sterilisation it will set firmly and be ready for use. Among the advantages that are claimed for this serum agar-agar are the following: (1.) It is quickly prepared, easily obtainable, and cheap; (2.) its selective action on the diphtheria bacillus is greater than that of any other serum preparation known to its authors; its inhibitory action on staphylococci bacillus, coli communis, etc., is extraordinary.

Examination of Cultures. The culture tube may be inoculated from a piece of membrane, if that be obtainable, first washing the

Saprogenic Cocci and Diphtheritic Bacilli.



FIG. 1.



FIG. 2.

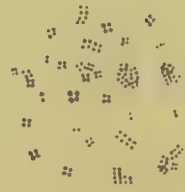


FIG. 3.



FIG. 4.



FIG. 5.



FIG. 6.



FIG. 7.



FIG. 8.



FIG. 9.

- FIG. 1.—Streptococci. FIG. 2.—Staphylococci. FIG. 3.—Micrococcus tetragonus.
 FIG. 4.—Film preparation, showing bacillus diphtheriæ. (Gentian violet.)
 FIG. 5.—Neisser's double stained diphtheria culture.
 FIG. 6.—Typical diphtheria bacilli, mostly of the long variety, only a few being short. (Agar streak, methylene blue stain.)
 FIG. 7.—Involution forms of diphtheria bacillus.
 FIG. 8.—Typical diphtheria bacilli. FIG. 9.—Pseudo-diphtheria bacilli (Hoffmann's bacillus).
 Magnified about 800 diameters.

membrane several times in sterile normal salt solution, and picking off a small piece with a platinum needle (previously sterilised by holding it in a flame till it is red-hot, and then letting it cool), or the platinum needle may be plunged into the substance of the membrane; or a sterilised needle or swab may be passed over the surface of the membrane in the throat. Then withdrawing the wool plug of the culture tube, the needle or swab is lightly drawn over the surface of the culture medium in the tube without breaking the surface, and the tube closed with the wool, which must not have been laid down, or it will have become impregnated with extraneous micro-organisms.

If no membrane is visible, or if the case is merely presumably a laryngeal diphtheria, the secretions from the fauces or the back of the pharynx should be examined—not that from the tonsillar surface.

Suspected milk or urine, etc., should be diluted, and then centrifugalised, cultures, or inoculations into guinea-pigs, being made from the sediment. The effect of dilution of the fluid is to lower its specific gravity, so that the bacilli tend to fall instead of permeating the fluid indifferently.

The culture tube having been inoculated, is then incubated at a uniform temperature of $38^{\circ}0'$ to $38^{\circ}5'$ Cent. In from twelve to eighteen hours it is ready for examination.

The macroscopical appearance of the growth is usually fairly characteristic. The Klebs-Loeffler bacilli at first form small, round, discrete, greyish-white spots, with a thicker and more opaque and slightly yellow centre; but the translucent spots by extension coalesce, and come to form an opaque patch, with a thick and prominent centre.

The bacilli may be stained by Gram's method with Loeffler's methylene-blue, but Max Neisser's double-stain is probably the most certain means of differentiating the diphtheria bacillus. Leedham-Green, who introduced the method in this country, speaking from a long experience, insists on the necessity for adhering strictly to Neisser's directions, viz., that the culture be grown in solidified blood serum at a temperature of 35° Cent. (certainly not higher than 36° Cent.), and that the growth should be examined when from 10 to 20 hours old. The bacilli are treated first with a dilute acid solution of methylene-blue, and subsequently with Bismarck brown. The stains must be freshly prepared, or the blue quickly loses its power. If examined too early the staining may be limited to only a few bacilli; on the other hand, if older than 20 hours, other bacilli beside the diphtheria bacillus may show a somewhat similar staining. Symes, of Bristol, my colleague at the Bristol Royal Infirmary, also speaks enthusiastically of the value, rapidity, and general trustworthiness of this method. The polar granules take

up the blue, and appear as dark blue or almost black dots, more oval than round, at one or both ends of the bacillus. Occasionally there is a third situated towards the middle, and sometimes a fourth is seen.

Kober insists on the importance of making the examination of the cultures after six hours, considering that the development of the Klebs-Loeffler bacillus is then at its height, and the majority typical in shape, whereas later on they assume a variety of atypical forms; moreover, at this early period the pseudo-bacilli are comparatively undeveloped.

The production of acid by the bacillus when grown in broth, generally, though not always, differentiates the true from the false bacillus. Both forms flourish in antitoxic serum.

Value of the Culture Method.—Apart from inoculation of guinea-pigs, the culture constitutes the most reliable diagnostic criterion we have; nevertheless, one bacteriological examination is not sufficient to negative the clinical diagnosis of diphtheria, however skilfully the inoculation, incubation, and microscopical examination are conducted, and several negative separate examinations must be obtained before a case presenting clinical evidence in favour of diphtheria can be safely excluded from that category.

But negative results should not be considered to outweigh decided clinical evidence in favour of a diagnosis of diphtheria, at any rate until several different cultures have been made yielding uniformly negative evidence of a case being one of diphtheria.

Inoculation of Guinea-pigs forms a reliable test, but is seldom resorted to except for the purpose of determining the virulence of a culture, and then, of course, only by those holding the necessary licence. Two or three minims of a blood-serum culture may be emulsified with sterile broth, and inoculated under the skin of the abdomen. With virulent cultures the animal dies in from thirty hours to seven days; spreading gelatinous exudation at the seat of inoculation, serous effusions in the peritoneum, pleural cavity, and pericardium, and hæmorrhage into the adrenals, are usual and characteristic features. The guinea-pig is very susceptible to diphtheria, and therefore it is probably practically safe to regard a culture which is non-virulent to guinea-pigs as harmless in man, the more so as there is some reason to doubt that virulence to guinea-pigs is an absolute criterion of virulence to human beings.

Pathological Anatomy.—At the outset diphtheria is undoubtedly a local lesion, due to the growth of the Klebs-Loeffler bacillus in or on the epithelium of the mucous membrane, but at first pro-

ducing only signs of simple inflammation. The earliest change is hyperæmia of the mucosa, with cloudy swelling of the epithelial cells. The diphtheria bacilli are present in greatest abundance in the more superficial granular parts of the exudation, but they are found in the epithelium and epithelial layers. The superficial layers of epithelium soon become necrosed, the dead epithelial cells being enclosed, together with the bacilli and leucocytes, in the meshes of fibrinous exudation.

The view till recently held, that the bacilli are found only in the neighbourhood of the affected area, is not absolutely correct, for they have been found in fatal cases in the lungs, spleen, and kidneys, etc.; yet they are probably generally limited to the local lesion, and are always most numerous there. The bacilli elaborate a toxin which, being absorbed in the circulation, produce the characteristic symptoms of the disease. Cultures from the blood of those dying early in diphtheria are usually sterile, but when the lung shows patches of broncho-pneumonia, diph-

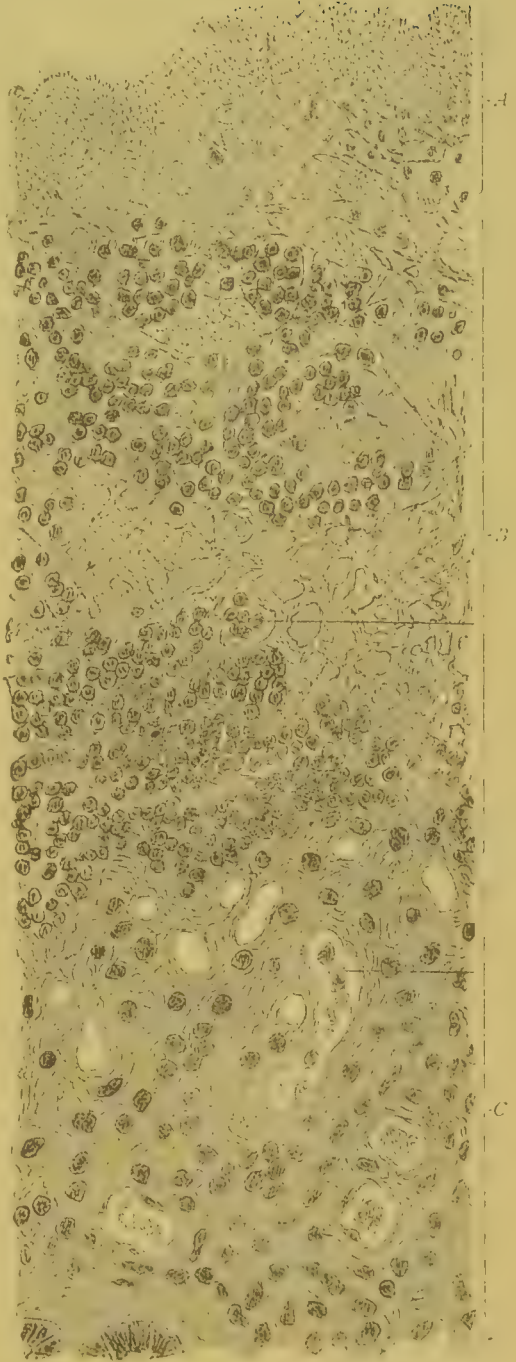


FIG. 65.

Surface of Diphtheritic Larynx. *A*, deposit of *D. bacillus* on surface of false membrane; *B*, false membrane; *C*, mucosa, the surface of which, at junction of *B* and *C*, is deprived of epithelium, some of which is seen lying in the lymph-vessels below. $\times 350$ diams. (Hamilton's "Pathology," Vol. II, Part I.)

theria bacilli are generally present in the blood. The toxin is a powerful poison, attacking especially the nerves; the heart, lungs, kidneys, and lymphatic glands also being affected.

As regards the nervous lesions, it would appear that certain slight and temporary changes occur in the nerve cells during the first twenty-four hours. The affected cells may either recover, or they may atrophy, with consequent degenerative changes of a Wallerian type and paralysis. Some of the cells recovering may account for the paralysis being usually incomplete. The paralysis is associated with the parenchymatous degeneration of the myelin sheath affecting both sensory and motor elements.

The heart is affected very early, and paralysis of the heart may occur early or late. The blood undergoes certain changes, the most important being a progressive leucocytosis, which subsides during convalescence.

The lungs may be affected with patches of broncho pneumonia, and the bacilli of diphtheria have frequently been found in these patches, but in almost all cases other organisms, such as streptococci and staphylococci, are found there also, and the broncho-pneumonia is probably due to these cocci. The kidneys are usually pale and swollen, and epithelial degeneration, and necrosis and nephritis may be produced, the inflammatory changes being most pronounced in the glomeruli and tubules. The urine and the kidneys then contain virulent Klebs-Loeffler bacilli.

Albuminuria occurs in from 24 to 60 per cent. of the cases, but actual nephritis is probably rare, and occurs in about 2 per cent. only.

The spleen is frequently enlarged, partly from vascular engorgement and partly from proliferative changes in the parenchyma.

Symptoms.—*Prodromal.*—Diphtheria being primarily a local infection, the initial symptoms are essentially local. In a few cases a sense of lassitude, aching in the limbs and back, and chilliness, accompanied by some rise of temperature and general febrile disturbance, precede the throat symptoms by a few hours, or even rarely a few days.

Local symptoms in the fauces, consisting in slight or considerable soreness especially on swallowing, cough and other symptoms of acute pharyngitis, and pains in the neck are usually the

first indications of the onset of an attack of pharyngeal diphtheria. If the fauces are examined at this early stage they will be found hyperæmic, swollen, and glazed, while the glands at the angles of the lower jaw may have begun to swell."

After an interval varying between a few hours and a few days, a false membrane generally (though not always) appears in one or more patches on the tonsils and uvula, or it may begin in small specks on the margins of crypts of the tonsil or on the uvula, spreading more or less rapidly, till, by their coalescence, these small patches, at first thin and translucent, become thicker and unite to form a characteristic tough, opaque, greyish-white false membrane, which may completely cover the uvula, soft palate and tonsils, and extend to the pharyngeal walls. Sometimes the patches remain discrete, so as to closely resemble acute follicular tonsillitis,—punctiform diphtheria. The small white patches are on the surfaces of the tonsil, not in the crypts, and cannot be squeezed out. Moreover, some will generally be found to present a rugged edge, unlike the accumulation within a tonsillar crypt. Yet other cases are precisely similar in appearance to simple acute lacunar tonsillitis, and it would baffle the acutest diagnostician to differentiate the one from the other without a bacteriological examination.

Marked rapidity in formation and the extent of the false membrane is some indication of the virulence of the disease, but is no reliable criterion. The pellicle is generally firmly adherent to the underlying mucosa, and on its removal a raw, ragged, and bleeding surface is left.

In some cases no pellicle is formed at any period in the attack, and these are by no means always mild. In others the membrane, instead of being thick, tough, and adherent, is thin, or friable, flocculent, and simply lying free, or so lightly attached to the surface of the mucous membrane that it can be readily removed by lightly brushing, leaving a slightly abraded or almost normal surface. Again, instead of being white or greyish-white in colour, the membrane may be a dark ash-grey or blackish or greenish grey, while copious muco-purulent discharge; with a foul odour, collects in the throat—the so-called putrid sore throat of older writers.

Speaking generally, the firm, whitish-grey membranes are almost pure cultures of the diphtheria bacillus, while the dark

grey, yellowish, or black, and the flocculent friable membranes yield mixed cultures of the specific bacillus, together with various pyococci and saprophytic micro-organisms. Opinions are divided as to the influence of mixed growths in the course of the attack. I am inclined to regard a mixed culture showing copious growth of pyococci (on a medium unsuited to their growth and examined within twenty-four hours) as distinctly unfavourable, for, though there are scarcely any cases in which prolonged culture and careful examination do not show their presence in at any rate small numbers, most hæmorrhagic and septic complications of diphtheria are due to their action, which the use of antitoxin cannot control. It probably depends on the initial virulence of the cocci whether they exert a pernicious influence, and increase the tendency to septic complications.

The lymphatic glands at the angles of the jaws and in the neck become swollen and tender, but rarely suppurate. In malignant cases the swelling is considerable, and extends beyond the glands, so that the whole of the neck, from the chin to the upper part of the chest, may be enormously swollen. The membranous inflammation may extend to the larynx, and down the trachea and bronchi, or upwards to the nose, giving rise to special features that are discussed elsewhere (see pp. 135 and 138). In rare cases false membranes have been found in the stomach and on the Peyer's patches in the intestines.

General. At the onset the temperature generally rises, but seldom exceeds 103° Fahr. or 104° Fahr., often falling slightly on the second or third day, and continuing moderately febrile for a few days longer. In very mild cases and in the severer forms the temperature is generally lower, and may be nearly normal, or in the worst cases subnormal, owing to the depressing influence of the poison in the circulation. The pulse, at first increased in frequency, and of the usual febrile type, tends to become weak and small, especially in severe cases. The heart's action is correspondingly impaired, and early heart failure is apt to occur in malignant diphtheria, the heart sounds becoming more or less indistinct or inaudible. The onset of symptoms of cardiac weakness may be gradual or may set in suddenly, and in the worst cases sudden death may occur during the first few days, or the patient may gradually sink and become unconscious after remaining several days in a more or less algid state. Pri-

mary heart failure is most likely to occur between the fifth and the fourteenth day, but may arise many days later.

The general symptom never absent, though varying greatly in degree, is prostration of strength. This feature may not be obvious within the first few days; on the other hand, it may develop so rapidly, and in such marked degree, that the patient may succumb to the depressing effect of the poison on the nervous system before the false membrane has had time to form. Rapid and pronounced anæmia and leucocytosis are usually observable, and in bad or fatal cases hæmorrhages from the mucous membranes, and a general cyanotic tint of the lips and surface of the body may appear, apart from dyspnœa. Various rashes of short duration occur in diphtheria, *e.g.*, large patches of erythema resembling scarlatina, or a vesicular and urticarial rash may occur—these are without significance; but ecchymoses, indicative of septic poisoning, are of grave import. The tongue is furred and the appetite lost. The spleen is usually enlarged.

The urine frequently contains albumin, which is very variable in amount. Much less often acute nephritis manifests itself by the presence of blood and casts in the urine. Various other complications and sequelæ may arise, apart from extension of the disease to the larynx or nose, *etc.*, *viz.*, lobular pneumonia, suppuration in the glands of the neck and in the middle ear, septicæmia, and post-diphtheritic paralyses.

Diphtherial Paralysis is liable to occur at a very early date, but may be delayed in its appearance two or three months. The paralysis is generally peripheral, but in rare cases cerebral hemiplegia has occurred.

Rarely the paralytic symptoms seem to precede instead of following the throat affection, and in these it is probable that the real cause of the paralysis is a previous unrecognised diphtheritic affection, of which the later throat affection is a relapse.

The onset of the paralysis is generally about two weeks after the subsidence of the primary attack, and the most common early symptoms are due to paralysis of the soft palate, *viz.*, regurgitation of fluids through the nose on swallowing and nasal voice. But other manifestations of nerve injury, *e.g.*, weakness of the arms or legs, facial paralysis, squint, paralysis of accommodation, of the diaphragm or the heart, may be the first indications of diphtheritic paralysis developing.

The muscles involved are chiefly those of the soft palate, of the eyeball, of the lower extremities, and of the heart. Miller has recorded 494 cases observed by him at the South-Eastern Hospital during the years 1896-1897, and of these 185 were primary paralysis of the palate, 197 strabismus, 10 paralysis of other muscles, and 102 cases of cardiac paralysis, of which only 11 did not end fatally. The bulk of the palatal paralysis occurred between the fifth and the fifteenth day, none before the fourth. Of the oculo-motor paralysis, the bulk occurred between the fourth and seventeenth days, none before the fourth; of the primary paralysis of other parts, half of them occurred between the tenth and fourteenth days, and none before the tenth day. A case of primary paralysis of the palate occurred as late as the sixty-fifth day; one of primary oculo-motor paralysis on the ninety-first day. The bulk of the cardiac paralyse occurred between the fifth and tenth days; a few cases occurred even as early as the second day, whilst this condition occurred in a fatal form in two cases on the fifty-fourth day, and in one case with subsequent recovery on the fifty-ninth day.

Diphtheritic paralysis is usually gradual in onset, but varies in the rapidity of its development and in severity and range. Speaking generally, the earlier the paralysis develops the more likely is it to be severe.

Paralysis of the limbs generally affects the legs before the arms, the arms often escaping altogether, the paralysis causing dragging of a leg or weakness of an arm, but rarely amounting to inability to use the limbs. Laryngeal paralysis may occur, and then affects the abductors sooner than the adductors of the cords. Anaesthesia of the larynx may be associated with the motor paralysis, and may lead to the escape of fluids and food particles into the trachea owing to the absence of a laryngeal cough reflex.

Paralysis of the diaphragm is associated with epigastric retraction during inspiration and distension during expiration (*i.e.*, the converse of the normal respiratory movements), and the respiratory excursions of the lower ribs are usually exaggerated.

Attacks of sudden onset and of alarming character are liable to occur, with symptoms pointing to disturbance of the cardiac and respiratory centres in the medulla. These are described by Guthrie as "bulbar crises." The chief features are rapid, weak pulse, running up to 150 or even 200 a minute, and syncope, urgent dyspnoea with shallow, irregular respirations, cyanosis and sweating. The cardiac and respiratory symptoms may occur together; in other cases one or the other set of symptoms is most marked. These bulbar crises are often accompanied by vomiting. Patients who develop these crises generally succumb.

Post-diphtheritic hemiplegias are exceeding rare, but may be due either to hæmorrhage or embolism.

Course.—In cases which do not succumb the membrane ceases to form after a variable interval, and it then soon disappears, being either shed in large or small shreds. or, becoming more diffuent, is lost in the secretions of the fauces. The mucous membrane is left swollen and red, but soon becomes normal. In severe cases the swelling of the fauces and palate may be very great indeed, and this may take some days to subside; in others superficial, or even deep sloughing ulcerations of the tonsils or fauces, or perforation of the palate, may occur. Relapses are rare, but sometimes arise before the patient recovers completely, a fresh membrane being deposited after the disappearance of that first formed, with recurrence of the usual symptoms; and though a relapse is seldom so severe as a first attack, it may be followed by fatal results.

A recurrent, pellicular sore throat, with the formation of false membrane, may occur for months after recovery.

Chronic Diphtheria.—In very rare instances it would appear that a chronic exudate may follow an acute attack, constituting a chronic faucial diphtheria analogous to chronic diphtheria in the nose. The persistence of Klebs-Loeffler bacilli in the fauces is sometimes long continued, and it may cause considerable inconvenience, for though the bacilli are generally attenuated in virulence, the patient cannot be pronounced safe to mix with healthy individuals so long as the specific bacillus is present and is virulent to guinea-pigs.

As a result of numerous "repeat" examinations, Hewlett and Nolan find that the bacilli are commonly to be found in the throat two to three weeks after the attack—sometimes, however, as long as seven, nine, and, in one case, twenty-three weeks after convalescence; and in the latter case their virulence, as tested by inoculation of the guinea-pig, was maintained to the last. Another notable case reported by Hewlett and Nolan was that of a school-boy who, six months previously, had suffered from an illness which was evidently diphtheritic, and from whose throat they obtained virulent Klebs-Loeffler bacilli; in all probability this case was the focus of infection from which originated a small outbreak of diphtheria among his class associates. Macgregor found virulent bacilli in a boy's throat nearly six months after an attack of diphtheria, and Schäfer records an instance where, seven and a half months after an attack, virulent bacilli were present. Bigg states that out of 405 cases of true diphtheria the bacilli disappeared within three days after complete separation of false membrane in 245 cases, not until seven days in 103 cases, twelve days in 34 cases, fifteen days in 16 cases, three weeks in 4 cases, and five weeks in 3 cases. Golay records an instance of apparent persistence of the Loeffler bacillus for 362

days in a boy aged five and a half years. The boy suffered from diphtheria in March, and up to August virulent bacilli were present in the throat. In September, in October, and in the following February he had relapses, with virulent bacilli in the throat. After this the parents would not consent to further examination as the child kept well.

The Prognosis depends on so many factors that it is very difficult to make definite statements on the point. Undoubtedly the age of the patient is an important element, the affection being very fatal in children under five years of age, and up to the age of fifteen the mortality remains high. The rate of mortality is highest during the first five years of life, especially between the ages of two and five; the period between five and ten comes next, and then that between ten and fifteen.

Another very important point is the duration of the disease before the patient comes under proper treatment. The statistics of the results of antitoxin injections show conclusively that with each day that has elapsed the mortality rate ascends, and, though in less degree, the same was observed in pre-antitoxin days. Sporadic cases are less often of a malignant type than those occurring in early days of a severe epidemic, and during the course of an epidemic there is a tendency for the virulence of the cases to decline.

Rapid extension of the membranes, when associated with marked fœtor of the breath, dark grey colour of the membrane, intense swelling of the glands and tissues of the neck, extension of the membrane to the larynx or nasal passages, obstinate vomiting, cardiac weakness, and a depressed temperature are all signs of bad import; but extensive membranous formation in the fauces is not *per se* of much prognostic value.

Vomiting is a very serious feature in some cases, and Hector Mackenzie has shown by statistics that vomiting coming on in the early and acuter stages is of far graver import than when occurring later.

Paralyses, except cardiac paralysis and post-diphtheritic hemiplegia, are not usually dangerous, and there is no proportion between the severity of the primary diphtheritic attack and their occurrence, while recovery from the paralysis is generally complete.

Treatment.—General treatment should be directed: (a.) To prevent the inoculation of other individuals by careful isolation, scrupulous cleanliness, and disinfection of all articles that may

have been contaminated ; (*b*,) To maintain the strength of the patient by avoiding all unnecessary exertion, by the frequent administration of light, easily assimilated food, and small quantities of alcoholic stimulants ; *c*,) To combat the general, and especially the cardiac depression, by the internal administration of such remedies as iron, quinine, strychnine, and strophanthus, and by the early and free administration of the antitoxin, to inhibit the action of the diphtheria toxin in the tissues ; and local treatment is necessary, (*d*,) To prevent the extension of the membrane and to destroy the vitality of the specific bacilli so as to prevent formation and absorption of the poison ; (*e*,) To overcome the obstruction to respiration when the membrane has extended to the larynx.

Serum Treatment.—It may be fairly said that diphtheria has been largely robbed of its terrors since the introduction of the antitoxic serum, and there are very few practitioners who have any doubt of its enormous value in controlling the disease, and if given early enough, completely changing for the better the clinical course of the great majority of cases. It is essential that the antitoxin be administered as early as possible, its efficacy in controlling the disease and reducing the mortality decreasing with each day that elapses between the onset of the disease and the commencement of the injections. This is the almost universal experience, a fact which demonstrates very forcibly the value of the antitoxin and the limitations of its usefulness.

The dose of antitoxin must not be too small. An average amount for an adult is 8,000 units in the first twelve hours, and after that from 3,000 to 2,000 units daily until the membrane disappears. If the attack appears to be particularly virulent, or if the case be not brought under treatment before the third day it is desirable to increase the initial dosage to 12,000 units, followed by 2,000 to 8,000 units every twelve hours for forty-eight hours ; whereas, in mild cases seen quite early in the attack, smaller amounts may prove equally effective. It is better to err on the side of an overdose, inasmuch as the antitoxin itself is practically harmless, though certain complications may arise from throwing considerable amounts of animal serum into the circulation, wherefore a serum of high potency is better than one containing relatively few antitoxic units per cubic centigramme. It is probably better to divide the larger initial doses ;

and, in attempting to prolong the period of immunity, it is better to repeat the dose rather than to increase its strength indefinitely.

In grave cases with urgent symptoms the serum may be given by intravenous injection apparently with good results, judging by three cases recorded by Gagnoni, all in young children with impending asphyxia.

Fisch has demonstrated by experiments on animals, on himself, and on friends, that artificial immunity may be induced by the administration by the mouth of antitoxic serum, or of milk from a cow or a goat artificially immunised to a high degree. But it takes from twenty-four to thirty-six hours for the complete absorption and diffusion of the antitoxin through the body, for even in eleven hours it did not render the blood serum antitoxic to an appreciable degree.

The effect of antitoxin upon the incidence of those complications which commonly occur in diphtheria is to cause a decided increase in the cases of albuminuria and paralysis, and these facts demand further consideration.

The Action of Antitoxic Serum on the Blood Corpuseles has been investigated by J. S. Billings, jun., Kanthack and Lloyd, and others. They found that the antitoxin treatment of diphtheria has no deleterious effects upon the blood corpuscles, but that, on the contrary, it seems to prevent degenerative changes that otherwise occur in the course of this disease, Billings more especially noting that the diminution of red corpuscles is much less marked than in those cases treated without it.

The Connection between Antitoxic Serum and Albuminuria.—Personally I have never been able to trace any connection between the serum injections and nephritis, but certainly it does appear to cause transient albuminuria. After the injections there occurs generally a slight transitory albuminuria and albumosuria; this was found by Siegert not only in patients already suffering from diphtheria, but also in healthy children in whom the antitoxin was injected as a prophylactic measure. If albuminuria be already present in a case of diphtheria the injection of antitoxin generally causes the albuminuria to disappear soon without evil consequences.

In patients actually suffering from nephritis when the serum is injected, the renal inflammation is often in no way aggravated, but may even be ameliorated.

Rash.—The two principal forms of rash due to serum injections are : (1,) Erythema ; and (2,) Urticaria. In rare cases joint pains and swellings have been noted. Morbilliform rashes are not uncommon, and a deep purple staining of the skin has been known to occur.

Effect of Antitoxin on Paralysis.—There is no room for doubting that with the administration of antitoxic serum the incidence of paralysis following diphtheria has increased. But the reason of this increased liability to paralysis in antitoxin cases is that many severe cases which would have died before the onset of paralysis, after antitoxin injections live to develop the nerve lesions, as is proved by the fact that if serum injections are given early enough, the number of cases of paralysis are lower instead of higher.

Local Treatment.—The local treatment that in my experience has proved most satisfactory is frequent spraying with a 1 in 1,000 solution of biniodide of mercury, in water and glycerin, equal parts. The mouth and fauces should first be cleansed with a 3 per cent. solution of Condyl's fluid, using a coarse spray. I have also employed with good results an acid, 30 vol. solution of the peroxide of hydrogen (Marchand's hydrozone), with 10 per cent. lactic acid, as it cleanses and disinfects the parts very effectually, and the acid inhibits the growth of the Klebs-Loeffler bacillus. The acidity may be disguised by adding sugar, and the transient pain it produces can be obviated by previous application of a weak solution of cocaine or eucaine. A fine spray should be used, as it insures the perchloride solution reaching every part of the fauces. Some prefer painting the parts thoroughly. The process of cleansing and spraying should be repeated every three hours, till the membrane disappears ; then once or twice daily for some days, carefully noting the reappearance of any fresh membrane as an indication for more frequent use of the germicide. It is necessary to watch for any indications of mercurial poisoning—a rare event. Insufflations of iodoform, or flowers of sulphur, have also been most serviceable. Loeffler has found the following mixture distributed over a surface, kills the diphtheria germs in twenty seconds, viz.: Alcohol and turpentine equal parts, with 2 per cent. of carbolic acid. A preparation even more speedy in action, giving the best results, is : Alcohol, 64 parts ; benzole (benzene) or toluol

(toluene) 36 parts; and 4 per cent. of iron-chloride solution. In dealing with sensitive patients and children, Loeffler recommends the addition of 10 per cent. menthol. He prepares his solution as follows: Menthol, 10 grammes; toluene, q.s. to make 36 c.c.; then add—creolin 2 c.c., iron chloride solution (sesqui-chloride) 4 c.c., alcohol absolute, q.s. to make 100 c.c. In applying, the mucus is first wiped off, and then a pledget of wool soaked in the solution is pressed forcibly against the diphtheritic membrane for ten seconds, and this application must then be immediately repeated. The applications are to be continued every three hours for four or five days, when all the local symptoms will probably have vanished. Unfortunately this solution does not appear to act so speedily on staphylococci and streptococci as on diphtheria bacilli.

It is generally considered undesirable to forcibly remove the false membrane, inasmuch as it quickly reforms, but if the pellicle can be easily removed with a brush or forceps, it is well to do so. Otherwise, its solution and disintegration may be accelerated by a steam spray of carbonate of soda. Papain and zymine have been advocated as solvents. The combination of papain and lactic acid (Formula 48) is especially useful.

Lactic acid solution (50 per cent. and upwards) may be carefully applied to the membrane, acting both as a solvent and disinfectant. Liquor ferri perchloridi, iodine (40 per cent.), chloride of zinc (gr. x to ʒj), chloral, and sulphurous acid, are suitable applications.

The galvano-cautery, and all powerful caustics and strong boracic acid, should be avoided. The temptation to push topical treatment is natural *a priori*, and was strongly advocated by Trousseau and his school, but is now generally abandoned, for it increases the local inflammation, wearies the patient, and is useless for the simple reason that the active bacilli invade the deeper structures of the mucous membrane, where these local applications can have little or no effect.

LARYNGEAL DIPHTHERIA.

(DIPHTHERITIC CROUP.)

Diphtheria in the fauces and pharynx in children is very prone to extend to the larynx, but only very rarely does the larynx become involved in adults.

Laryngeal diphtheria is rarely primary. Thus Northrup reports that in one hundred and fifty-one cases of diphtheria, in only one was the deposit confined to the larynx. As in faucial diphtheria, cases of laryngeal diphtheria occur in which no false membrane can be discovered either *intra vitam* or post mortem.

The **Symptoms** of laryngeal diphtheria are at the outset almost indistinguishable from those of membranous croup, being mainly hoarseness, with a loud ringing cough, soon passing into aphonia with a hoarse stridulous or "croupy" cough. The temperature as a rule is lower than in membranous croup, being only slightly above normal, while the early presence of albumen in the urine and marked constitutional depression should be regarded as evidence of diphtheria.

An examination of the larynx, if obtainable, may reveal the greyish false membrane lying on the slightly hyperæmic mucous membrane, and in adults this is seldom difficult, as dyspnœa is not generally present. But in children it is often impossible for any but an expert to get a view of the larynx without a dangerously prolonged and exhausting struggle, and moreover the fact that no membrane can be seen proves nothing. It is only rarely that children will expectorate false membrane before tracheotomy has been performed, although membrane is generally coughed out after tracheotomy. It therefore becomes necessary to rely on other symptoms in children for arriving at a diagnosis, and these are: (1.) Alterations in the voice; and (2.) Signs of laryngeal obstruction. The vocal signs are hoarseness or aphonia, with a loud ringing or a hoarse cough.

Laryngeal obstruction is indicated by early laryngeal stridor and later by dyspnœa, and, in young children, by recession of the epigastrium and lower ribs on inspiration, and by the *pulsus paradoxus*, i.e., absence of the radial pulse during inspiration.

Since the laryngeal affection is almost invariably associated with, and secondary to faucial diphtheria, there is seldom any room for doubt as to its real nature; but every case of membranous laryngitis must be assumed to be true diphtheria until repeated bacteriological examination proves the contrary. The danger is that the absence of more acute signs of laryngeal obstruction, owing to the great depression of the vital powers, should lead one to overlook, or under-estimate, the evidence of extension of the membrane to the larynx.

These symptoms of pronounced dyspnœa may not develop for the first two or three days; in other cases they supervene rapidly, and in any case urgent dyspnœa may suddenly develop at any time, either from displacement of a piece of false membrane obstructing the small glottic aperture, or from inflammatory infiltration in the submucosa.

The course of the disease is marked by progressive enfeeblement of the patient as in faucial diphtheria, but more especially by a tendency to pulmonary engorgement and collapse.

Occasionally the false membrane is expectorated *en masse*, and the symptoms of laryngeal obstruction and dyspnœa subside for the time being, but generally soon return. More usually in cases which proceed to recovery the symptoms of laryngeal obstruction and dyspnœa gradually decline, and in the course of recovery the various special complications and sequelæ of diphtheria may appear. But the mortality is very high, especially in young or weakly children, the majority of deaths being directly due to asphyxia from laryngeal obstruction or extension of the membrane into the trachea and large and small bronchi, together with wide-spread bronchial catarrh, pulmonary engorgement, lobular pneumonia and collapse. Increased feverishness may mark the supervention of the lung lesions, but the progressive lividity may be accompanied by diminished subjective dyspnœa. Patients who do not succumb to these conditions may recover, but many are carried off from cardiac failure or from general progressive debility passing into drowsiness, loss of consciousness, and death.

Prognosis.—The prognosis depends chiefly on: (*a*,) The patient being brought under treatment early; (*b*,) The age of the patient. In no class of cases has the effect of antitoxic serum in reducing mortality been more clearly demonstrated than in laryngeal diphtheria. The chief cause of death being the laryngeal obstruction with resulting pulmonary complications, the younger the patient the more grave is the prognosis, owing firstly to the relative, as well as absolute, smallness of the glottis, and secondly to the yielding of the chest walls in young children. Thus a patient without marked laryngeal obstruction, or marked evidence of general debility, and treated during the first two days of illness with antitoxic serum, has a very fair chance of recovery; but when marked obstructive

dyspnœa has persisted for several hours, pulmonary complications will probably cause death if the patient is under five years of age, even if tracheotomy or intubation be performed.

Diminution in the respiratory embarrassment, associated with increasing lividity, and with a small weak pulse and low temperature, is an ominous sign, as it implies extensive pulmonary lesions with greatly diminished vital power.

Treatment.—Everything that has been said on the treatment of faucial diphtheria applies equally to diphtheria of the larynx, trachea, and bronchi, excepting the local application of germicides, which is hardly feasible. But the patient should be surrounded by warm, moist air, preferably a steam bed. Steam sprays impregnated with carbonate of soda may be inhaled with advantage every two or three hours. Emetics should generally be avoided; although, with a view to getting rid of false membranes, it is sometimes desirable to administer ipecacuanha to the point of vomiting when the bronchial tubes have become blocked with secretion.

Antitoxic serum should be given early and freely in the same doses as for faucial diphtheria. The percentage of cases requiring intubation or tracheotomy is thereby greatly diminished, while the mortality of both intubated and tracheotomised cases has been reduced enormously. A fairly average table of results are those recorded by Martin and Hunt of cases at the University College Hospital, London:—

The percentage mortality in four non-antitoxin years was	70, 68, 78, 47
“ “ “ three succeeding antitoxin years (1895-96-97) was ...	33·3, 40, 23·5
“ “ “ of 169 intubated or tracheotomised cases in four non-antitoxin years was ...	65·5
“ “ “ of 72 intubated or tracheotomised cases in three antitoxin years was ...	26·4

Intubation and Tracheotomy.—When obstructive dyspnœa is urgent, or even if not very urgent yet persistent for several hours, the question of intubation or tracheotomy demands immediate consideration, and on no account should such measures be delayed with (*a*), increasing cyanosis, (*b*), marked inspiratory recession at the epigastrium, (*c*), the absence of a radial pulse during inspiration. Opinion is divided as to the advisability of early operation. Laryngeal diphtheria is extremely fatal:

firstly, because such an extensive deposit is sure to involve more rapid absorption of the toxin, especially as the deposit is more or less beyond the reach of effective local germicides ; secondly, because the membrane rarely stops at the larynx, but extends down the trachea and bronchi ; thirdly, because it may cause death by closure of the glottis. Therefore, unless the dyspnœa is really marked, the operation is very often altogether useless, and one hesitates to advise an operation which cannot prevent the death of a patient from heart failure, or from blocking of the bronchi.

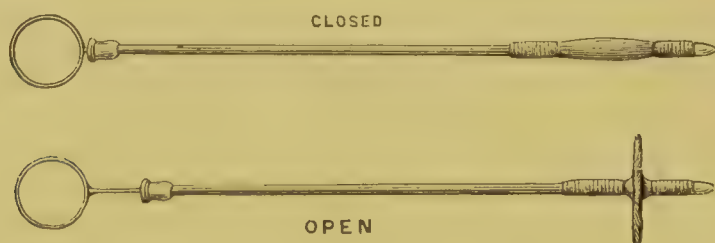


FIG. 66.
Tracheal Probang as used at the Bristol Royal Infirmary (Half-size).

On the other hand, the performance of tracheotomy greatly facilitates effective local treatment of the infra-glottic extension of false membrane, and we may therefore be enabled to prevent further extension, and, in a few cases, pull the patient through. Speaking generally, under all other circumstances, I should prefer intubation as soon as dyspnœa supervenes. If tracheotomy has been done, the local treatment should be carried out through the tracheotomy wound. If the membrane is extending down the trachea, it should be removed as far as possible by forceps, or by the tracheal probang, and small quantities of the perchloride solution may be injected through the tracheotomy wound. (See also "Intubation," chap. ix.)

RHINAL DIPHTHERIA.

The essential pathology of rhinal diphtheria differs in no respect from diphtheria of the fauces, the presence of the Klebs-Loeffler bacillus differentiating this form of rhinitis from all others.

Symptoms.—Acute rhinal diphtheria is nearly always secondary to the throat affection, and in most patients extension of the disease from the pharynx is indicated by nasal obstruction and a copious discharge of acrid muco-pus, sometimes streaked with blood or at ended with epistaxis. The discharge is very

irritating to the skin of the nasal orifice and upper lip, causing redness and excoriations around the anterior nares on which false membrane may form. Perhaps owing to the rapid absorption of the toxin by the vascular mucosa, it is an extremely fatal complication ; but there is some reason to believe that only very virulent Klebs-Loeffler bacilli will flourish in the nasal passages and produce marked symptoms, and this may explain the usual gravity of nasal symptoms in diphtheria. It is well to remember that paresis of the soft palate causes an escape of fluid food by the nose, which might be mistaken for nasal discharge.

Primary diphtherial rhinitis is generally "latent" and unattended by any general symptoms, being a purely local affection characterised by the formation of false membrane in the nasal passages, and consequent nasal obstruction. Lambert Lack states that diphtheritic rhinitis (which he terms fibrinous rhinitis) is so common that the cases amounted to $2\frac{1}{2}$ per cent. of all the children attending his hospital practice. The absence of marked constitutional disturbance is certainly not due to the diphtheria bacilli being non-virulent, for in a large proportion of cases the membrane yields a pure culture of typical long Klebs-Loeffler bacilli which prove highly virulent on inoculation of guinea-pigs, and in this fact lies the great danger of an apparently benign, purely local, nasal trouble, superficially resembling nasal catarrh, being the cause of diphtheria in those who are exposed to contagion. An instructive, but by no means isolated, instance in point occurred in Clifton, and is recorded in a report of the Medical Officer for Bristol, D. S. Davies : " A patient of a medical man suffered from an indefinite form of sore throat, and three weeks later the doctor's two children attended a party at the lady's house and were fondled by her. At intervals of eleven and sixteen days the children developed nasal membrane *without other disturbance of health*, and, portions of the membranes having been found by Dowson to contain the true bacillus, material was also submitted to Klein, who also found the disease to be diphtheria of a virulent type when tested by inoculation of guinea-pigs. Active local antiseptic treatment had been employed for some two months, when the children were declared practically free from the disease. But at the end of other two months their mother sickened with a disease which also proved to be true diphtheria, and the bacillus was again recovered from

the children's nasal membrane. The malady in the mother ran a mild course, and at the end of another month no bacilli could be recovered from any one of the three patients."

Another variety of diphtheria is the external diphtheritic rhinitis described by Todd as occurring in children convalescent from scarlatina. The first sign is the appearance of a slight redness at the posterior margin of one or both nostrils. The redness becomes more intense, and ultimately a moist granular-looking raw surface results. Todd summarises his observations thus: (1,) Children convalescent from scarlet fever in hospital are very liable to a certain form of external rhinitis, often accompanied by the formation of secondary pustules on various parts of the body; (2,) This rhinitis, though not membranous, is associated with the presence of the Klebs-Loeffler bacillus in the nostrils, this organism being absent from the fauces; (3,) It is contagious as such; but has not been observed to give rise to faucial or laryngeal diphtheria; (4,) It is unaccompanied by rise of temperature, albuminuria, or marked glandular enlargement; (5,) It appears to be limited to children under thirteen years of age, and has been most frequently observed at the ages of three and four years. The fact that the bacillus, though present in the nostrils in large numbers and causing a local lesion, does not give rise to any constitutional symptoms or to faucial or laryngeal diphtheria, suggests that its virulence is modified to a remarkable extent.

In nasal diphtheria a warm solution of salt in water (5j to the pint), followed by perchloride of mercury solution (1 in 3000—4000) should be used three or four times daily with the patient lying on one side. The nozzle of the douche is gently placed in the nostril and the stream allowed to flow in slowly, returning by the other nostril.

CHAPTER VI.

CHRONIC INFECTIVE DISEASES.

SYPHILIS—TUBERCULOSIS—LUPUS OF THE PHARYNX AND LARYNX—
MYCOSIS.

SYPHILIS.

Inherited Syphilis, affecting the pharynx, usually manifests itself in early infancy or at the age of puberty, and may assume the form of either secondary or tertiary lesions.

Primary Sore.—Though rare, this has been observed in a good many cases, chiefly on the tonsils; very occasionally on the faucial pillars. "Pain is slight, the tonsil is enlarged and the surface red. Generally there is no marked ulceration; but some erosion is always present, with a well-defined and a sharply-cut margin, although seldom much elevated. The base is generally covered with a slight, whitish, sticky secretion. On palpation there is marked induration, and often stony hardness. The submaxillary glands are enlarged in all cases" (BulkleyDuncan). The nature of the sore is easily mistaken when, as is often the case, it covers the whole tonsil. The well-marked bubo (which seldom or never suppurates), associated with a recent sore throat, should lead to the suspicion of syphilis, which the development of secondary symptoms confirms.

The local treatment consists in prescribing a mercurial or other antiseptic gargle.

SECONDARY SYPHILIS.

Erythema gives rise to discomfort rather than pain. It presents a peculiar, almost characteristic bright, bluish-red, symmetrical hyperæmia, not extending beyond the soft palate, with an almost sharply defined border. This appearance should always lead to the suspicion of syphilis, evidence of which is generally present in other secondary phenomena.

Mucous Patches.—These more or less bilaterally symmetrical, slightly elevated, bluish-white patches on the tonsils, faucial pillars and pharynx, closely resemble the appearance produced by the application of lunar caustic to mucous membrane, and are surrounded by an erythematous blush. The general symptoms enable one to distinguish this form from diphtheria.

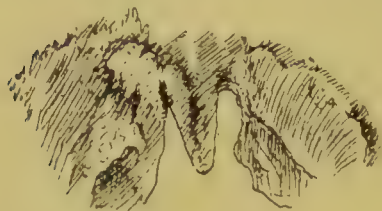


FIG. 67.
Secondary syphilis.

Superficial Ulcerations usually present the peculiar bilateral "Dutch garden symmetry" (J. Hutchinson), so characteristic of secondary syphilitic lesions. They are prone to occur on both tonsils, but the ulceration may be confined to the posterior surface of the soft palate.

TERTIARY SYPHILIS.

Gummas are rarely seen without ulceration, since syphilitic deposits do not give rise to much pain and ulceration occurs rapidly. A gumma may arise in the soft palate, in the tonsil, or in the posterior pharyngeal wall, as a smooth or uneven swelling, covered with somewhat congested mucous membrane, which soon shows yellowish spots indicative of commencing necrosis.

Tertiary Syphilitic Ulceration is always due to the disintegration of a gumma. In early cases these ulcers are found mostly in the soft palate, faucial pillars and uvula. In ulceration occurring many years after the initial lesion they are more often seen on the tonsils and posterior wall of the pharynx as well. The extent and the depth of the ulcerative process varies greatly, since the amount of gummatous deposit is so variable.

While both superficial and deep syphilitic ulcers are sufficiently obvious if they occur on the anterior surface of the soft palate and fauces, etc., the inexperienced observer often fails to detect the ulceration when confined to the posterior surface of the soft palate and uvula, and to the rhino-pharynx. An intensely injected boggy-looking infiltration of the velum or uvula should put us on our guard, and lead to the inspection of the posterior surface of the soft palate and the rhino-pharynx by the rhinoscope. Sometimes an extensive ulceration here

extends down to the margin of the velum and can be seen on anterior inspection.

Tertiary syphilitic ulceration is often remarkably rapid, and the tissues affected may be extensively disintegrated before the diseased process can be arrested (see *Plate XI*). The importance of not overlooking the real nature of the case is therefore obvious, since a few days' delay may entail a large perforation of the velum or its complete destruction by an ulceration extending from the posterior surface. Phagedenic ulceration, which is very difficult to arrest, occasionally occurs, especially in those much broken in health and in chronic alcoholic patients.

CICATRICAL LESIONS.

Deep syphilitic ulceration is generally followed by distortion and contraction of the tissues involved, and not only is the soft palate often completely destroyed, but the destructive process may have involved the loss of the hard palate and floor of the nasal passages. The cicatrices are usually stellate or radiating in appearance, owing to the contraction of the original site of the ulcer.

When the destruction is less extensive the pillars of the fauces are often much contracted, and adhesions between the soft palate and the posterior pharyngeal wall may have produced almost complete occlusion of the rhino-pharynx.

Diagnosis.—(See p. 159.)

Treatment.—(See Syphilis of the Larynx.)

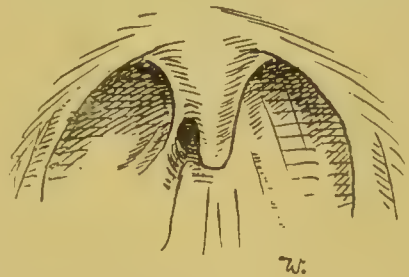


FIG. 68.

Cicatricial adhesion of the uvula to the posterior wall of the pharynx, due to tertiary syphilis.

TUBERCULOSIS.

Etiology and Pathology.—Tubercular disease of the pharynx and fauces may be either primary or secondary, but it is almost invariably secondary to tubercular disease of the lung.

Two forms occur, the acute and the chronic, the latter comprising the vast majority of cases. Only two or three cases are recorded where the acute form was said to be primary:—

(a.) Secondary or acute miliary tuberculosis of the pharynx

is due to diffuse infection of the submucous lymphatics from some pre-existing tuberculous focus.

(b,) Localised tuberculous deposits, which generally soon disintegrate with the formation of irregular indolent ulceration, are probably due to primary local infection through the surface of the mucous membrane.

Symptoms.—The *acute form* usually commences by the onset of pain in the fauces, which are found to be slightly swollen, and at first hyperæmic. The soft palate, if the seat of the deposit, becomes stiff and paretic, and in the course of a day or two several discrete, muddy-grey tubercles are visible, slightly elevated, but obviously beneath the translucent mucous membrane. In from one to three days the tubercles coalesce and begin to ulcerate, fresh miliary tubercles in the meanwhile appearing in the pale mucous membrane, only to pass through similarly rapid phases of development. The paretic, stiffened soft palate fails to shut off the naso-pharynx on deglutition, even before ulceration has produced extensive disintegration; wherefore the voice is nasal, and fluids run into the nose in drinking. Deglutition and coughing are generally very painful; consequently the patient is unable to get rid of the copious, sticky, stringy, muco-purulent discharge covering the parts, and can only make feeble attempts at hawking.

As in acute miliary tuberculosis affecting the lungs, there is elevation of temperature, but the emaciation and general prostration are more rapid. The cervical glands soon become enlarged, but seldom very painful or tender.

The *chronic form* is decidedly rare, though probably many cases are overlooked. The relative frequency of tuberculous tonsils, which clinically resemble ordinary chronic hypertrophy, is referred to in the section on "Diseases of the Tonsils." In primary tubercular disease of the pharynx, the deposit usually appears along the posterior pillars of the fauces, and resembles an aggregation of large bluish-red, lymphoid granules, but they are tender and hard to the touch. When they disintegrate the ulceration is indolent, and granulations and nodular thickening may cause it to resemble lupus. The cervical glands may soon become enlarged and tender. Perforation of the soft palate very rarely occurs, and the appearance of perforation should lead to the suspicion of syphilis.

Tuberculosis of the Pharynx.



FIG. 1.



FIG. 2.

FIG. 1.—Primary tuberculosis of the pharynx.

FIG. 2.—Secondary, or acute miliary tuberculosis of the pharynx.

For these drawings the author is indebted to Dr. Walter F. Chappell, of New York.

By the kindness of Dr. Chappell of New York I am enabled to present excellent examples of secondary and primary pharyngeal tuberculosis (see *Plate XIII, Figs. 1 and 2*).

The tonsils are occasionally the seat of tubercular deposit, either alone or in association with the fauces. In a case reported by Lublinski, the right tonsil was enlarged and greatly congested, and there were five ulcers on this and two on the left tonsil, varying in size from the head of a pin to a lentil, whose bases were covered with a whitish detritus, the margins of the ulcers being only slightly raised, but somewhat redder than the surrounding parts. The shape of the ulcers was in no way characteristic; the larger ones were somewhat oval.

A remarkable clinical fact in both forms of the disease, to which Chappell directs attention, is the tendency for the right side of the pharynx to be first invaded; this he attributes to the difference in the arrangement and distribution of the lymphatics on the right and left sides of the neck.

The **Diagnosis** of the acute form has to be made from diphtheria, follicular tonsillitis, syphilis, herpes and small-pox, while the chronic form must be distinguished from lupus and syphilis.

The **Prognosis** of the acute form is exceedingly grave, the disease almost invariably ending in death in from two to six months. In the chronic form, the disease is more indolent, and may heal under local treatment.

Treatment.—The local treatment of the acute form is mainly palliative and consists in sucking ice, spraying the throat with a 4 per cent. solution of cocaine, or 20 per cent. menthol in liquid vaseline, or the insufflation of powdered boracic acid containing gr. $\frac{1}{8}$ of hydrochlorate of morphine to each insufflation. If not very acute, the daily application of a solution of lactic acid (20 to 80 per cent.) to the whole of the ulcerated surface may be beneficial.

See also "Treatment of Laryngeal Tuberculosis," p. 200, the same measures being indicated in Chronic Pharyngeal Tuberculosis.

LUPUS OF THE PHARYNX AND LARYNX.

Lupus of the nose and throat is generally regarded as a rare affection, but it is often present without giving rise to symptoms sufficiently definite to attract the patient's attention. Laryngeal

lupus is generally secondary to lupus in the pharynx or the skin ; not very rarely it is primary.

Middlemas Hunt finds that in a collection of four hundred and eleven cases of external lupus, no less than 20 per cent. were affected either in the pharynx, larynx, or nose. In one hundred and seventy-three cases of lupus of the mucous membranes occurring in the clinic of Doutrelepon, only six were free from cutaneous manifestations of the disease, while of these one hundred and seventy-three cases, seventy-five were affected in the nose, thirty-one on the palate, and uvula, and thirteen in the larynx.

It occurs with very much greater frequency in females than in males, and generally reveals itself at, or before, puberty.

Etiology.—As regards the etiology of lupus there is no doubt that the disease is directly due to a specific bacillus ; but while the great majority of authorities are agreed in regarding lupus and tuberculosis as one and the same disease manifesting itself under different conditions, their identity is denied by others, and it cannot be said that their pathological identity has been conclusively demonstrated, while their very different clinical manifestations favour the view that lupus and tuberculosis are pathological as well as clinical entities.

Symptoms.—Lupus vulgaris develops very slowly and insidiously, for, as already stated, it is often present in the pharynx and larynx without manifesting any definite symptoms. The patients generally complain of some stiffness in the pharynx, or of soreness and tickling sensation, and in some cases of slight dysphagia. If the larynx be involved, the symptoms are chiefly impairment of the voice or dyspnoea. Pain is generally absent or only slight.

Physical Signs.—The characteristic aspects of lupus of the pharynx and fauces are well shown in the coloured figures that Prof. Chiari, of Vienna, has very kindly allowed me to introduce. When the deposit first manifests itself on the uvula, or the free border of the soft palate, we may find localised tumefaction, generally of distinctly heightened colour, less marked, and more limited, than in syphilis or acute pharyngitis, but differing from the anæmia premonitory of tubercle ; and sometimes the deposit is in apparently healthy mucous membrane. In course of time, smooth, hard nodules appear, varying in size from a pin head

Lupus of the Palate and Larynx.



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 5.

FIG. 1.—Lupus of the hard and soft palate and of the fauces, showing cicatrices, disseminated nodules, and ridges of the tubercles.

FIG. 2.—Earlier stage of hyperæmia and slight swelling. The epiglottis partly eaten away by lupus ulceration.

FIG. 3.—Lupus of the larynx, showing very similar condition. Lupus tubercles on the left vocal cord.

FIG. 4.—Lupus of the larynx. At the base of the tongue and on the swollen epiglottis are seen the characteristic tubercles and ulcers; tubercles are present also on the left ventricular band.

FIG. 5.—Lupus of the larynx. The epiglottis has partly disappeared; a typical nodule is seen in the right vocal cord.

The author is indebted to Professors O. Chiari and Riehl, of Vienna, for permission to reproduce the first four drawings. The remaining figure from a case under the author.

to a split pea or larger, of a more distinctly rosy colour. The nodular deposit soon produces a considerable and somewhat characteristic twisted and distorted appearance of the parts, and the uvula is often remarkably elongated as well as thickened.

After a variable period the nodules become soft and apple-jelly like, and ulcerate, or occasionally a nodule is absorbed without ulceration. The ulcers present a serpiginous, worm-eaten appearance, with defined, hard, or soft margin, granular and prominent, and with velvety, red, dry, indolent base. The process of ulceration and cicatrization is very slowly progressive, with periods of increased activity alternating with lengthy periods during which the disease appears stationary. In this manner the whole of the uvula and soft palate may be lost, and the disease may spread to the hard palate. When the tonsil is affected it becomes covered with irregular red nodules and pits of ulceration.

In the larynx the disease usually first attacks the free margin of the epiglottis, which shows unilateral tumefaction, gradually extending to the ary-epiglottic folds and ventricular bands. The vocal cords are generally the last to be affected; and so slow is the progress of the disease that they often escape, but when they are involved they become red and unevenly tumefied. When the nodules appear on the epiglottis they pass through the same stages as in the palate; but in the ulcerative stage the epiglottic appearance is characteristic, becoming quite pale, worm-eaten, and rough, or large portions of it are ulcerated away. The infiltrated ary-epiglottic folds are pale, contracted and tumefied. When cicatrization has caused a stenosis of the pharynx, dysphagia becomes more marked, and if the soft palate has been eaten away the voice is nasal, and fluids find their way into the nose in deglutition.

When the larynx is first attacked, there may be no symptom to attract attention; but when the disease has extended to the arytenoids and posterior commissure, the voice becomes impaired or lost, while the contraction of the tissues, together with the thickening and the nodular deposits, may occasion such a degree of stenosis that tracheotomy has to be performed. There is never any inflammatory exudation in lupus, and never perichondritis or necrosis of laryngeal cartilages.

Diagnosis.—The diagnosis has to be made from simple chronic pharyngitis, syphilis, cancer, and tuberculosis.

In the absence of cutaneous lupus, the difficulty in excluding syphilis, acquired or hereditary, is considerable, and we are often obliged to wait for the result of anti-syphilitic treatment to prove negative. I have alluded to the main distinctions between lupus and syphilis; and the fact that lupus may occur in the very young, is very slowly progressive, always with cicatrisation, and is almost never painful, together with the characteristic appearances of the growth, and the absence of wasting, fever, or quickened pulse, should rarely leave us in doubt as to the diagnosis. (See also p. 159.)

The **Prognosis** as regards life is most favourable, the only real danger being stenosis of the larynx; but that comes on gradually, and is not very liable to be suddenly increased by perichondritis or œdema, so that tracheotomy can almost always be performed in good time. Haslund records one death from asphyxia.

Treatment.—As regards local treatment, the nodules and tumefactions should be scarified or curetted, and strong lactic acid rubbed in by the methods employed in laryngeal tubercular disease (p. 200). This should be done once a week, successive portions being treated until the whole of the diseased area has become cicatrised and no nodules or ulcers are visible. The case must be watched for a year at least after apparent cure has been effected, and any fresh manifestations must be similarly dealt with. If only tumefaction is present, linear scarification, followed by the application of lactic acid and glycerin, equal parts, or two to one, is recommended by De la Sota.

Isolated deposits may be destroyed by the galvano-cautery. Mandl's solution of iodine, solutions of nitrate of silver, chromic acid solution, and 1 in 1,000 solution of perchloride of mercury, are also recommended as local applications.

I have seen very great improvement characterised by very marked diminution in the tumefaction and ulceration follow repeated injections of minute doses of tuberculin. My practice has always been to employ doses just insufficient to cause pronounced local reaction.

Excellent results are said to follow treatment by the X-rays,

and also with Finsen's concentrated light method. I have seen apparent improvement under the X-rays, but both this and the light method are at present only in the stage of "trial," and it must suffice to simply mention them here as procedures worthy of fuller investigation.

Stenosis of the larynx may be arrested for a time by intubation, or by dilatation with Schrötter's bougies.

MYCOSIS.

(PHARYNGOMYCOSIS LEPTOTHRICIA.)

Etiology and Pathology.—The leptothrix fungus is universally present in tartar, decayed teeth, and in the crypts of the tonsils. Under certain conditions it takes root and grows in the mucous membrane, and constitutes the affection *pharyngomycosis leptothricia*. Many varieties of leptothrix are met with, just as there are many varieties or different species comprised under the generic term of streptothrix actinomycotica.

Symptoms.—It occurs in two forms, the diffuse and circumscribed. In the diffuse form, milky patches are seen usually on the dorsum of the tongue. The more circumscribed form most frequently arises in the tonsillar crypts, from which firmly adherent chalk-white horny excrescences are seen to project. They are also seen at the base of the tongue, on the fauces and uvula, and on the posterior pharyngeal wall, and the affection has been observed in the nasal passages.

The symptoms are always either very slight, or altogether absent. Patients may complain of discomfort, stiffness, and dryness in the throat, and the growth may weaken and impair the voice. They are generally run down and in poor health.

Diagnosis.—The fact that the surrounding mucous membrane is healthy, the growths firmly adherent and white, and that there is a complete absence of pain or constitutional disturbance, should prevent any error in diagnosis, while a microscopical examination will show the characteristic thread-like cryptogam in the midst of the amorphous granular matter.

Treatment.—The eradication of the fungous growth often gives considerable trouble, and usually local treatment is singularly unsuccessful. The chief point is to improve the general health of the patient, and in most cases the mycotic

trouble will disappear spontaneously. Scraping and the use of the galvano-cautery may be successful when all other measures have failed.

Solutions of bichloride of mercury (1 in 1,000), applied locally, in combination with a gargle of the same (1 in 2,000), are favourably commended (Chiari), and others have found good results follow the application of an alcoholic solution of salicylic acid.

Simple removal of the growth is useless, as it invariably grows afresh.

CHAPTER VII.

NEOPLASMS AND NEUROSES OF THE PHARYNX.

BENIGN NEOPLASMS—MALIGNANT NEOPLASMS—DIFFERENTIAL DIAGNOSIS
OF SYPHILITIC, TUBERCULAR, LUPOUS, AND MALIGNANT ULCERS—
SENSORY NEUROSES—MOTOR NEUROSES.

BENIGN NEOPLASMS.

BENIGN growths in the fauces are not at all common, yet almost every kind and sort of tumour is met with.

Papilloma is by far the most common form of benign tumour in this region, the small, warty, sessile or pedunculated, light pink growths, with granular or cauliflower-like surface, being usually attached to the margin of the soft palate, the pillars of the fauces, or to the uvula. They often give rise to no symptoms, unless they attain considerable dimensions. Less frequently *adenoma* occurs as a hard, rounded, sessile growth of slow development in the palate or tonsil; *fibroma* and *angioma* are of very rare occurrence in the fauces. *Fibroma* forms a rounded, hard, smooth, rapidly growing, red tumour, and is liable to bleed freely. *Angioma* appears as a purplish, soft, highly vascular growth with irregular surface, and is sometimes pedunculated.

Symptoms are chiefly due to mechanical interference with the action of the soft palate and pillars of the fauces, and to difficulty in swallowing, altered phonation, etc. Fibromas are sometimes painful. A small warty epithelioma might easily be mistaken for a papilloma, but it would be associated with hyperæmia of the tissues around.

Calcareous concretions occur in the tonsil, and rarely in the soft palate, and may simulate a growth.

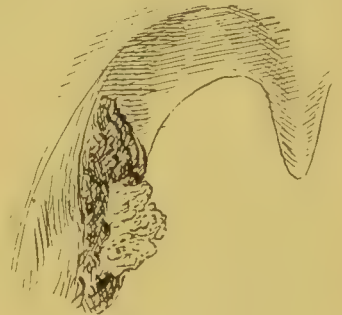


FIG. 69.
Papilloma of the tonsil.

Treatment.—A papilloma should be cut off, the tissues immediately around the base being included in the excised growth. The other benign growths should be removed only when their presence causes inconvenience or pain.

MALIGNANT NEOPLASMS.

Growths of the rhino-pharynx are discussed in the section on diseases of the nose, and are excluded from consideration here.

Etiology and Pathology.—Malignant disease of the fauces and pharynx is fairly common, sarcoma occurring at any age, carcinoma only very rarely before the age of forty. The causes of malignant growths here, as in other parts of the body, are still open questions, but heredity and local irritation appear to have some influence. The morbid anatomy of these growths needs no description here, since it differs in no respect from similar growths in other regions.

Sarcoma of the pharynx occurs in the form of lympho-sarcoma, round-celled, spindle-celled, mixed round and spindle-celled, alveolar, melanotic, and myxo-sarcoma, of which the first-mentioned is the most common. A possible pathological connection between lympho-sarcoma of the fauces and Hodgkin's disease is suggested by Butlin, and Kundrat has noted two cases of lympho-sarcoma in leukaemia; on the other hand, Chiari states that lympho-sarcoma differs from leukaemia and pseudo-leukaemia by the absence of enlargement of the lymphatic glands in other regions, and enlargement of the spleen and liver.

Two noteworthy facts are, (1,) that malignant disease of the pharynx is nearly always primary, or due to direct extension from neighbouring structures—very rarely is a "metastatic" secondary deposit seen here, and (2,) the secondary glandular enlargement usually occurs very soon, and is very pronounced.

Symptoms.—In the earlier stages the symptoms of malignant disease of the fauces and pharynx are often most indefinite; in many cases, indeed, the first thing to attract the attention of the patient is the secondary glandular enlargement behind the angle of the lower jaw. In other cases it is the difficulty in swallowing

which is first complained of, especially in growths arising in the laryngo-pharynx.

But in carcinoma gradually increasing pain is seldom long delayed, the pain being especially noticed on swallowing, of lancinating character, darting up to the ears. Salivation is another early symptom. When ulceration occurs the pain is severe, the breath becomes foetid, the difficulty in swallowing and the glandular enlargement rapidly increase, and cachectic symptoms develop rapidly, and the patient usually succumbs in from six to twelve months at the outside from the commencement of secondary glandular infiltrations.

Sarcomas differ greatly in their degree of malignancy and rapidity of development, but the chief symptoms are those of mechanical interference with deglutition and speech. Pain is seldom a marked feature, at any rate until the growth ulcerates, and then foul muco-purulent discharge, pain and loss of appetite, and generally increasing weakness are almost invariably present. Hæmorrhage is often severe, but the ulceration, as a rule, is not deep.

Objective signs.—Carcinoma usually originates in the lateral aspects of the oro-pharynx, in the pillars of the fauces, less often in the tonsil, soft palate or uvula, while in the lower pharynx it often spreads very soon to the larynx, or if arising from the posterior wall, it overhangs and partly conceals the posterior portion of the larynx. The growth has a pink, granular, uneven surface, and soon ulcerates, and in the earlier stages has often a wart-like aspect, surrounded by a hyperæmic infiltration. It grows rapidly and extends in various directions. When ulceration has occurred, the floor of the ulcer is ragged and covered with foul *débris* and muco-pus, the margins being defined and slightly elevated.

Lympho-sarcoma, according to Chiari, appears either as a definite tumour on the tonsil, or as an infiltrating growth which soon ulcerates. The larger growths or flat aggregation of small tumours may break down, and the resulting ulcers heal partly or entirely, so that even deep scars may be formed, only to be followed by fresh deposit of marrow-like nodules, or thickening.

A remarkable instance of the spontaneous disappearance of a lympho-sarcomatous tumour in a patient at the Bristol Royal

Infirmity is recorded by Munro Smith. The primary lesion appears to have been at the angle of the jaw. This was extirpated ; it recurred, and after an unsuccessful attempt at removal of the recurrent growth, the latter spontaneously disappeared. The patient finally succumbed to recurrent growths in other regions.

Sarcomas are generally succulent and bright red in aspect until ulceration occurs. The rate of growth is very variable, and in some cases it remains localised for a considerable period.

Diagnosis.—In *carcinoma* the neighbouring glands are generally enlarged and indurated; but this condition is by no means invariable, and the absence of obvious enlargement of the glands of the neck does not exclude the diagnosis of cancer. If involving the tonsil, digital exploration gives a characteristic fixed, indurated feeling; the tonsil soon ulcerates, and then in appearance closely resembles tertiary syphilitic disease. The diagnosis can hardly be made between these affections, till the failure to check the progressive ulceration by anti-syphilitic remedies leaves no doubt as to its real nature. Large doses of iodide of potassium will sometimes relieve the pain, but the malignant ulceration progresses unchecked. The most insidious, and by no means the least common, seat of deposit is the lower end of the pharynx, where the commencing growth can only be observed in profile, so that the very slightly elevated upper margin may alone be visible, just behind or above the posterior wall of the larynx.

Sarcoma of the fauces and tonsils is less hard than epithelioma, and varies very much in its rate of growth, some cases showing a tendency to remain localised for a considerable period. It spreads by extension to the neighbouring regions, and very generally involves the deeper tissues behind the angle of the jaw, so as to cause large swellings in the neck.

In both sarcoma and carcinoma the onset is often insidious; but cancer develops more rapidly than sarcoma, pain is more marked, ulceration occurs earlier, and the growth is hard, pale pink, or even bluish-pink, with a well-marked areola round the growth. The ulcers are deep, with hard raised margins, and are covered with yellowish-grey *débris*. Carcinoma of the tonsil occurs

Epithelioma of the Pharynx.

FIG. 1.

The palate is extensively involved, especially on the right side; the uvula is enormously enlarged and pendulous.

generally after the fortieth year. While enlarged tonsils dating from childhood may persist throughout life, an enlargement commencing in an adult, especially if unilateral, must always be regarded with grave suspicion.

A gumma often presents a close resemblance to sarcoma, but the syphilitic neoplasm is much more rapid in development, does not spread gradually so as to involve neighbouring structures, is usually unilateral, and is rapidly and favourably influenced by large doses of iodide of potassium.

Prognosis. — As spindle-celled lympho-sarcoma grows less rapidly than the round-celled form, occasionally remaining encapsuled for some time, when secondary extension is slow to appear we are justified in giving a *relatively* hopeful prognosis in the former class if they come under operation early.

These remarks may, perhaps, appear unduly favourable, and of course any form of malignant disease in the throat is a serious complaint; but many instances of the successful removal of these growths should warn one not to lightly dismiss the possibility of a cure in cases coming before us.

With regard to carcinoma the case is less hopeful; but with limited growths in accessible regions, and before the secondary glandular enlargement is extensive, the possibility of successful radical operation must be carefully weighed without loss of time. These are questions that can only be answered satisfactorily by an expert, who will have to consider (1,) the possibility of complete eradication; (2,) the risks involved; (3,) the functional results should the operation contemplated prove successful; (4,) the chances of recurrence.

We are greatly indebted to Watson Cheyne for having worked out the results of operations for malignant disease of the pharynx by various surgeons. He classifies the cases into five groups, according to the parts involved, viz., when the disease is (1,) in the soft palate, or at the upper part of the tonsillar region spreading on the palate; (2,) limited to the tonsillar region; (3,) at the lower part of this region, spreading on to the back of the tongue or the aryteno-epiglottic folds; (4,) in the pharynx, further back and lower down; and (5,) in the pharynx, spreading on to the orifice of the larynx. As a rule, enlarged lymphatic glands are present in all the cases, and require removal.

Of these groups the first is the most favourable for operation ; the risk is not great, and the functional result is good, though, if much of the soft palate has to be removed, the closure of the velum may be imperfect; likewise cancer limited to the tonsil, or to one or other of the faucial pillars, is favourable. But with cancer in the other regions the condition is very serious, and most serious of all are cases where the disease has not remained limited to the pharynx, but has spread on to the orifice of the larynx—the great immediate danger of operation in these cases being septic pneumonia; and this risk is greatly increased where the orifice of the larynx or the back part of the tongue is interfered with.

Finally, Cheyne refers to the enormous risk of operations involving the aryteno-epiglottic folds, and he thinks that if they are to be considered at all as regards operation, the only plan is to excise the larynx in addition to the diseased portion of the pharynx, a procedure which introduces the question of functional result.

When operation is out of the question, palliative measures are often necessary. Thus local applications of powdered orthoform, or insufflations of morphia will be useful in allaying pain when ulceration has occurred, and the use of various antiseptic gargles or sprays will serve to cleanse the foul surfaces. When deglutition becomes no longer possible, the advisability of performing gastrostomy will have to be considered, and with respiratory obstruction due to involvement of the larynx tracheotomy may likewise become necessary.

In conditions for which medicine and surgery can offer but little hope of relief, it is justifiable to resort to a procedure which has apparently yielded successful results in a few cases, viz., the injection of erysipelas and bacillus prodigiosus toxins according to the method of Coley for sarcoma.

DIFFERENTIAL DIAGNOSIS OF SYPHILIS, TUBERCULOSIS, LUPUS AND MALIGNANT DISEASES OF THE PHARYNX.

Syphilitic, tubercular, lupous and malignant ulceration in the nose, pharynx, and larynx present certain characteristics which, without being absolutely pathognomonic, are generally sufficiently definite to enable the practised eye to make a diagnosis

in any given case. It is necessary, however, to bear in mind that mistakes are occasionally made by the most experienced, and that we can never afford to dispense with the assistance of any facts in the family or personal history of a case which may aid us in making a diagnosis. Moreover it should not be forgotten that septic ulcers and ulceration of the pharynx in diabetes and other affections may closely resemble those due to syphilis or tubercle.

Primary Syphilis.—A chancre on the tonsil may be mistaken for tertiary ulceration, tuberculosis, or epithelioma. From tertiary ulceration it is distinguished by its superficial character, the stony hardness of the tonsil, and the large cervical bubo. Tuberculous ulcers are superficial, but with irregular, ill-defined margins. Epithelioma is less rapid in development, but a definite diagnosis may be impossible till secondary lesions appear and the effect of antisiphilitic treatment has been observed.

Mucous patches may superficially resemble a diphtheritic deposit, but a close inspection would show the difference between a mucous patch and false membrane, while the co-existence of other syphilitic phenomena would indicate the real nature of the suspicious plaque.

Gumma may be mistaken for *quinsy*, especially if pain and feverish symptoms are present; but the absence of marked tenderness and acute inflammation of the fauces generally would serve to distinguish it when the other evidences of syphilis are not observable. It may also be confused with fibroma, sarcoma, or epithelioma. The former is rare and of slower growth, while from the malignant growths gumma is distinguished by its inflammatory aspect and the effect of antisiphilitic treatment.

The Superficial Syphilitic Ulcer is definite, cup-shaped or with a flat base, with an even, slightly raised margin, surrounded by a well defined border of brightly injected mucous membrane. The floor of the ulcer is covered with whitish-yellow, sticky, disintegrating muco-purulent *débris*.

The Deep Syphilitic Ulcer is crater-like, with an undermined, slightly elevated, regular, sharply cut margin, surrounded by a well defined areola, with the base covered by yellowish ropy muco-pus and necrotic tissue. The ulceration advances more in

depth than in superficial extent. It is followed by cicatricial contraction which gives rise to great deformity.

Tubercular Ulcers present an uneven, ragged, "mouse-nibbled" margin, which is not elevated, surrounded by pale greyish mucous membrane. Ulceration extends superficially rather than deeply, the base being nearly flush with the surrounding swollen tissues; and being covered with greyish disintegrating tissue, it is often difficult to determine the exact limits of the ulcer, which progresses very slowly. In the earlier stages, the miliary tubercles, which have not broken down and coalesced to form the irregular ulcer, may frequently be seen.

Lupous Ulceration is invariably associated with the characteristic nodules, and spreads very slowly in one part while cicatrising in other directions, so that the active ulceration is rarely very extensive at any one time, whilst the mucous membrane surrounding the diseased process is, except for the venous injection, normal in hue. The affected area is always deep red or rose coloured, and the margin of the ulcer is either hard or soft, irregular, defined, and elevated. If ulceration has gone on sufficiently long, the characteristic cicatricial bands traverse the area previously occupied by the slowly advancing ulceration, yet without producing the marked distortion of old syphilitic ulceration. Recurrent ulceration of the cicatrix is pathognomonic of lupus (Chiari). The floor of the ulcer is depressed and crater-like, velvety and uneven in contour, and seldom presenting any *débris*, but is indolent and dry.

Malignant Ulceration is associated with a definite and generally increasing tumour, covered with bluish-pink or pale mucous membrane, and surrounded by diffuse injection and infiltration. The ulcer is deep, with well marked, slightly elevated nodular and rapidly advancing margin, the floor of the ulcer being irregular, thickly covered with the foul-smelling disintegrating tissues and muco-pus. Epithelioma often presents bluish-pink, indolent, cauliflower excrescences.

The subjoined table (*vide page 159*) of differential diagnostic signs briefly summarises the main points of distinction between some of the diseases of the tonsils, etc.

TABLE OF DIFFERENTIAL DIAGNOSTIC SIGNS.

CHANCRE.	SYPHILIS, 2NDRY & 3RY.	TUBERCULOUS ULCERATION.
<p><i>Functional Symptoms.</i>—The first symptom is a stinging pain in the tonsil, but with little pain on swallowing, which is never so difficult as in cancer or in tertiary syphilis.</p> <p>Cancer occurs in late middle life, but sarcoma may also occur in the young; chancre generally in young adults.</p> <p><i>Physical Signs.</i>—The surface is very red, but there is always a well defined erosion, with sharply cut margin, from the commencement. Induration or even stony hardness. The sub-maxillary glands early enlarged.</p> <p>Like cancer and tertiary syphilis, and unlike secondary, it is unilateral.</p> <p>No hæmorrhage, only streaks of blood.</p> <p>No emaciation, early appearance of secondary rash, etc.</p> <p>Most amenable to treatment.</p>	<p><i>Symptoms.</i>—Often no pain, or if present swallowing never intensely painful. Wasting and cachexia in proportion to the difficulty in taking nourishment, and not very pronounced. No salivation.</p> <p>In secondary syphilis of the tonsils and fauces there is generally bilateral deposit of mucous patches and superficial ulceration, with well marked bright red areola.</p> <p>In tertiary syphilis the tonsils are unilaterally affected by a deep perforating ulcer.</p> <p>The margins of the ulcer are often undermined and overhang the deep lying ulcer, the floor of which is covered with necrotic tissue.</p> <p>The sympathetic glandular enlargement is slight, and not painful as in cancer.</p> <p>Hæmorrhage slight or absent.</p> <p>The rapid improvement under antisyphilitic remedies is always a valuable sign</p>	<p><i>Symptoms.</i>—Swallowing is always very painful, and loss of flesh rapid, with nocturnal rise of temperature, and a general well marked tubercular cachexia is always present. There is early and rapid infiltration of the parts around, with very early tendency for fluids to return through the nose on swallowing.</p> <p><i>Physical Signs.</i>—General pallor, with diffuse infiltration of the affected region. Early superficial, irregular, mouse-nibbled ulceration, with grey debris. In the earlier stages the deposits of miliary tubercles are very characteristic; these ulcerate and coalesce. No inflammatory areola.</p> <p>Hæmorrhage generally absent.</p> <p>Usually concomitant disease of larynx and lungs.</p>
ACUTE TONSILLITIS.	CARCINOMA.	SARCOMA.
<p><i>Functional Symptoms.</i>—Pain very marked from the commencement, great tenderness and difficulty in swallowing. Generally some rise in temperature. In cancer, onset of pain is gradual.</p> <p><i>Physical Signs.</i>—Characteristic redness and inflammatory infiltration. Follicular exudation, but no ulceration. May proceed to suppuration.</p> <p><i>Chronic abscess</i> of the tonsil may be diagnosed by incision and discharge of pus.</p> <p>Most amenable to treatment.</p>	<p><i>Symptoms.</i>—Dysphagia is always an early symptom, and pain is considerable and persistent, but of gradual onset. Increased pain on swallowing becomes so great as to prevent the patient taking food.</p> <p>Saliva accumulates in the mouth.</p> <p>Early and well marked cachexia, and rapid loss of flesh.</p> <p><i>Physical Signs.</i>—Carcinoma always presents an enlargement, with superficial irregularity of surface, which is light pink or bluish, and soon ulcerates, with granular fissured surface, hard, elevated margin, general cartilaginous hardness and fixedness. Ulceration not very depressed, covered with foetid muco-pus.</p> <p>Early infiltration of neighbouring glands.</p> <p>Hæmorrhage frequent and often profuse, sometimes fatal.</p> <p>Generally unilateral.</p>	<p><i>Symptoms.</i>—Difficulty and pain in deglutition, sometimes very slight, and until ulceration occurs, is chiefly mechanical.</p> <p>Saliva accumulates and dribbles from the mouth.</p> <p>Loss of flesh generally rapid.</p> <p><i>Physical Signs.</i>—Sarcoma attains considerable dimensions before ulceration commences. The growth is red, fleshy looking, and soft, surrounded by a well marked bright red areola.</p> <p>Spreads to neighbouring regions and externally to the neck.—especially rapid is the extension of round-celled sarcomata.</p> <p>Hæmorrhage is frequent and sometimes fatal.</p> <p>Generally unilateral.</p>

SENSORY NEUROSES.

The fauces and soft palate derive their sensory nerves from the second division of the fifth (see *Plate XXXV*), and from the vagus and glosso-pharyngeal nerves. The pharynx is supplied by the glosso-pharyngeal, which is shown in *Plate XXXIV*, dividing into branches which enter the constrictors.

Anæsthesia, partial or complete, may be *bilateral*, *e.g.*, from neuritis after diphtheria, or in hysteria, bulbar paralysis, or insanity; or *unilateral*, *e.g.*, in the pharynx, from interference with one glosso-pharyngeal nerve by pressure of a tumour.

Hyperæsthesia, *Paræsthesia*, and *Neuralgia* are often met with in neurotic patients apart from any organic disease, and are also suggestive of gout, or may be premonitory of pulmonary tuberculosis.

Thus sensory neuroses are mainly of psychical origin, and occur in hysteria, at the climacteric, and in neurasthenia, melancholic or insane patients. Some of the more obscure neuroses, both in the male and female, have a sexual basis.

The physiological association between the upper respiratory tract and the sexual organs is seen in the developmental changes which occur in the male larynx at the time of puberty, coincident with the alteration known as "the break of the voice," and in females by the huskiness in the voice which many singers notice at the menstrual period. Wright has found obvious differences in the nasal mucosa of the bull and the bullock, and the modifying effect of castration in boys on the normal vocal development at puberty is well recognised. Violent sneezing sometimes occurs in the male on sexual excitement, and in the female well authenticated instances of vicarious bleeding from the nose or pharynx replacing menstruation have been recorded.

Comprised under the vague term "hysterical" we meet with numerous and very various functional neuroses in the nose and throat; and though these conditions are more common in chlorotic or anæmic females about the time of puberty, and also at the climacteric, they are by no means confined to one sex. *Anæsthesia*, *hyperæsthesia*, and *paræsthesia* and *neuralgia* of the pharynx are especially common at the climacteric, and often cause manifest distress to the patient; but the sufferers are not usually of the "hysterical" type. The distress complained of varies immensely; in one case it amounts to no more than inconve-

nience from constant tickling, hemming, or the sense of a hair or fish-bone in the throat, or a sense of heat or cold ; in others the pain is neuralgic in character, and is so severe that the patient dreads cancer or consumption. Often the sufferer has much difficulty in describing exactly what she does feel, or even to precisely localise the seat of the abnormal sensations, while general depression and emotional disturbance are almost usual. Similar affections occur in neurasthenic males, especially in the highly strung, whose health is impaired from excessive mental work.

The **diagnosis** of sensory functional neuroses rests mainly on the exclusion of all organic lesions, taken in conjunction with the history of the case and the general condition of the patient. The objective symptoms in sensory neuroses are either completely absent, or only such slight departures from the normal condition are present that it becomes extremely improbable that they can be held responsible for the symptoms. Chronic rhinopharyngeal catarrh, considerable anæmia of the pharynx, commencing tuberculous disease or cancer, and foreign bodies are among the chief *local* conditions, and organic nerve affections, toxic neuritis, gout, rheumatism, and dyspepsia the chief *general* conditions that may simulate functional neuroses and call for differential diagnosis.

Treatment.—Nervine tonics are indicated, and the treatment of any underlying dyscrasia, such as anæmia, gout, dyspepsia, and portal congestion. The climacteric cases are often very difficult to relieve, but the patient should be encouraged to look forward to spontaneous subsidence of the symptoms in course of time, and not permit herself to dwell too much on what is generally a transient neurosis. Faradisation or galvanisation may be useful in the post-diphtheritic cases, combined with the exhibition of strychnine either by the mouth or hypodermically. Gouty and dyspeptic patients are often benefited by a course at Carlsbad, Kissengen, Vichy, or Aix-les-Bains, etc.

Any local cause of irritation should be removed, and the general health should receive attention.

MOTOR NEUROSES.

PHARYNGEAL PARALYSIS.

Paralysis of the soft palate results from intracranial disease in which the vago-accessory nuclei or nerve fibres are involved,

as, for instance, in acute or chronic bulbar paralysis, locomotor ataxia, syringomyelia; or from peripheral nerve lesions, such as pressure by new growths or enlarged glands, gumma, or from neuritis due to diphtheria or other diseases; or the paralysis may be myopathic from inflammatory infiltration. Hughlings Jackson has drawn attention to the comparative frequency with which paralysis of the tongue, palate and vocal cords are associated, and also of the sterno-mastoid and trapezius in some of the cases. The number of cases of paralysis limited to these muscles points to a close anatomical connection between their bulbar nuclei of origin. Hysterical paralysis of the pharynx and œsophagus is occasionally met with. The view that paralysis of the soft palate is due to and accompanies paralysis of the facial nerve has now very few supporters.

Symptoms.—The accessory nerves may be involved either unilaterally or bilaterally. When the lesion is unilateral, the uvula is usually drawn towards the healthy side, and the velum is lower and less arched on the affected side. Sometimes the uvula hangs straight. If bilateral, the velum hangs loosely, and does not become elevated when locally stimulated. The voice is nasal, and fluids may escape into the nose during deglutition. As the paralytic condition of the pharyngeal constrictors becomes more marked, deglutition gets more and more difficult, and the difficulty in swallowing fluids is always greater than for solids. When the difficulty in swallowing is due to obstruction, it is naturally first noticed, and always more pronounced, in reference to solid food.

Diagnosis.—From the nature of the lesions that induce paralysis of the pharynx sensory neuroses are often conjoined. Neuropathic paralysis should be distinguished from mechanical impairment of the muscles concerned by new growths, cicatrices, etc. The dysphagia may give rise to the suspicion of mechanical obstruction of the œsophagus or lower portion of the pharynx by new growths, pressure of aneurysm or other tumours; but in mechanical obstruction the difficulty in swallowing is, of course, more pronounced for solids than for fluids.

PHARYNGEAL SPASM.

Spasmodic affections of the muscles of the pharynx and larynx occur in neurotic individuals and in hysteria, insanity,

chorea and tabes. The so-called "globus hystericus" is probably due to spasm of pharyngeal muscles.

Courmont records a case of tonic pharyngeal spasm in locomotor ataxia, and Kellogg relates the case of a man insane from alcoholic excess with clonic unilateral spasm of the right sterno-mastoid accompanied by a loud inarticulate noise; the noisy part of the phenomenon was regarded as an instance of (psychic) vocal tic engrafted on a spasmodic muscular tic. "Clonic lingual spasm and laryngeal spasms are not very rare symptoms in general paresis and tonic œsophageal spasm and pharyngeal spasm in hysterical insanity." Kellogg also refers to a kind of spasm, usually provoked in origin, met with in patients under the tension of persistent delusions or emotions, as in melancholia attonita and like states. When such a subject is urged to speak or otherwise aroused, the only response may be spasm of one or several muscles of the face, neck, or pharynx.

Spasm of the pharynx may occur in association with acute inflammatory affections, *e.g.*, acute tonsillitis. Clonic spasm of the levator palati gives rise to a peculiar sound audible to the patient and those around. The cause is in some cases obscure; but it is often a reflex neurosis, and therefore any possible source of irritation in the nose or naso-pharynx should be sought for and rectified.

Bernhardt had a female patient, aged thirty, who for several weeks had contractions of the whole velum palati, the palato-glossal and palato-pharyngeal arch, the posterior pharyngeal wall and the base of the tongue, the contractions being at the rate of 120 a minute, accompanied by a clicking sound. The vocal cords were unaffected. The patient was otherwise quite healthy, and displayed no neurotic symptoms. In a very similar case Semon observed clonic adductor movements of the vocal cords also.

Treatment.—In addition to general treatment, appropriate to the condition with which these motor neuroses are associated, the application of the faradic or galvanic current will often prove most efficacious in relieving spasm.

CHAPTER VIII.

*ACUTE AND CHRONIC INFLAMMATIONS OF
THE LARYNX.*

ACUTE LARYNGITIS—MEMBRANOUS LARYNGITIS—CHRONIC LARYNGITIS—
PACHYDERMIA LARYNGIS—CHORDITIS TUBEROSA—CONGENITAL LARYN-
GEAL STRIDOR—EDEMA—PERICHONDritis—DISEASES OF THE CRICO-
ARYTENOID JOINT.

ACUTE LARYNGITIS.

ACUTE catarrhal laryngitis in adults is a minor ailment that is sufficiently familiar to most people, and, except in professional voice users, often hardly attracts the serious notice of the patient. In children, however, owing to the anatomical peculiarities, it is frequently attended with the gravest symptoms.

Etiology and Pathology.—The usual *exciting* cause is exposure to damp, cold, or any sudden change in temperature; or it may be set up by inhaling steam or irritating vapours, etc., by the action of dust, or excessive straining of the voice. *Predisposing* causes are sedentary habits, especially in those who live in close and over-heated rooms or who indulge freely in alcoholic liquors, the rheumatic or gouty diatheses, and all forms of nasal obstruction producing mouth-breathing. It may arise during an attack of measles, influenza, typhoid fever, and more rarely in scarlet fever, and typhus. Frequent attacks of laryngitis often precede true laryngeal tuberculosis, and should lead one to examine the lungs, etc., for any indications of commencing phthisis.

The laryngeal mucous membrane is heightened in colour, and this alteration is most striking in the normally yellow epiglottis and white vocal cords. The superficial layer of epithelium is extensively shed, and in the parts subject to attrition the denudation of epithelium may be more complete, leading to the formation of superficial, symmetrically disposed ulcers on the

vocal processes, the interarytenoid fold, and the anterior commissure. In the milder forms, there is at most only slight thickening of the mucous membrane, and only a very slight secretion of viscid mucus on the vocal cords.

When the attack is more severe the submucosa becomes infiltrated, and the lower part of the epiglottis, the ary-epiglottic folds, and the loose mucous membrane in the inter-arytenoid fold may be obviously thickened.

Symptoms. — *In adults* the symptoms are purely local, or associated with those of a severe cold. There is hoarseness, or more or less complete aphonia, and a dry, tickling cough, with little or no expectoration, unless the inflammation extends to the trachea and bronchi, when the laryngeal symptoms will be associated with those due to tracheo-bronchitis.

If the mucous membrane is thickened from submucous infiltration the symptoms are aggravated, and, in addition to more marked constitutional disturbance, and a loud barking croupy cough, the voice is completely lost; moreover, from the resulting laryngeal obstruction there may be considerable inspiratory stridor, for though it is unusual to find marked symptoms of laryngeal obstruction in adults, the occasional supervention of acute œdema must not be forgotten. Even in the milder forms it is common to find imperfect approximation of the vocal cords on phonation from paresis of the internal thyro-arytenoidei muscles, or of the arytenoideus. Gerhardt affirms that the paresis of the thyro-arytenoidei in laryngitis is due to implication of the nerve endings, while Schrötter believes that it is due to inflammatory infiltration of the muscles; it is probably due to both these causes (see *Plate XVI, Fig. 1*).

Local variations in the character and distribution of the inflammatory changes have been distinguished by the terms *epiglottitis*, *arytenoiditis*, or *chorditis* when the epiglottis, arytenoid folds, or the vocal cords are respectively the parts mainly implicated; *l. hypoglottica*, when the submucosa below the true cords is œdematous; *l. hæmorrhagica*, when the local inflammation is attended with hæmorrhage (see also Laryngeal Hæmorrhage, p. 87); *l. herpetica*, when vesicles are present and associated with cutaneous or pharyngeal herpes; *l. sicca* is generally a chronic affection, but acute laryngitis without exudation has been described under this name.

In children acute laryngitis is generally productive of severer symptoms than in adults. Sappey has shown that in children the lymphatic supply of the mucous membranes is far more extensive than in adults, and Bosworth cites this fact as a possible explanation of the tendency for acute laryngitis in children to be attended with sub-glottic infiltration of the sub-mucosa. In childhood, too, the glottic opening is *relatively* (as well as absolutely) small, and the mucous membrane is more vascular and less firmly adherent to the underlying structures. Moreover, in children, paresis of the intrinsic muscles of the larynx is more readily induced by inflammatory infiltration than in older patients, and there is a greater tendency for reflex nerve phenomena, such as laryngeal spasm, to occur. Thus we have a sufficient explanation of the usually marked clinical difference between acute laryngitis in adults and in children. For these reasons, even simple catarrhal laryngitis in a child must not be regarded too lightly. Occurring in the first two or three years of life it is certainly a dangerous affection; and, as is the case in all inflammatory affections of the respiratory tract, the danger is more or less in inverse ratio to the age of the patient. The temperature is moderately febrile, 100°—101° F., the pulse quick and hard, and the respirations frequent and laboured. At first the cough is harsh, soon becoming loud and brassy, and usually there is some laryngeal spasm following the cough. In a short space of time muco-purulent expectoration becomes copious, and the cough is then less distinctly croupy in character. The symptoms are worse towards evening, but are not sudden in onset, nor is there the marked intermission in the laryngeal stridor that is always observed in spasmodic laryngitis.

SPASMODIC LARYNGITIS.

(Laryngitis stridula or false croup.)

This form of acute laryngitis must not be confused with the purely nervous affection *laryngismus stridulus* on the one hand, and true membranous croup on the other. In spasmodic laryngitis we have the symptoms of catarrhal laryngitis with intermittent laryngeal spasm and less catarrh. There is generally subglottic infiltration of the mucous membrane of the larynx, and one attack predisposes to another.

The usual history is that the child has caught a cold, and respiration is a little embarrassed towards evening. There is a dry hard cough, which, however, does not prevent it falling asleep. Towards midnight it suddenly awakes with a laryngeal spasm, respiration is greatly embarrassed, the cough is loud and brassy, with marked inspiratory stridor from laryngeal obstruction, which persists till death seems imminent from acute asphyxia. In a few minutes the spasm passes off, respiration becomes easier, and the child falls asleep; and though restless and disturbed by occasional croupy cough, the spasm may not recur again. Towards morning there is marked remission of the symptoms: but the following night or two the laryngeal spasm recurs, yet, almost invariably, with diminishing severity, the catarrhal symptoms and croupy cough persisting for some days.

(Contrast the symptoms with those of true croup, p. 169.)

Treatment. — In adults, simple acute laryngitis should be treated by absolute rest of the voice and confinement to a warm room. Free evacuation of the bowels, and abstinence from the use of tobacco and alcohol, should be enjoined. A cold compress may be applied externally to the laryngeal region, till all the acute symptoms have passed off. Ice may be sucked, or we may resort to steam inhalations containing compound tincture of benzoin, camphor, eucalyptus oil or creasote; or an atomiser, containing menthol with, or without, a small quantity of camphor, cocaine, or morphine, may be used for spraying into the larynx several times a day. Internally ipecacuanha and cubebs, or ammonium chloride, are of service.

When the acuter symptoms have passed off, more stimulating inhalations are often desirable, such as oil of Scotch pine, or the *pinus pumilio*, 5j to the pint of nearly boiling water, and the steam inhaled.

Professional singers and public speakers frequently require that, if possible, an attack of acute laryngitis should be aborted, or at least kept in abeyance sufficiently to enable them to keep some important engagement. In such cases the bowels should be freely evacuated by some saline aperient or calomel. Absolute rest of the voice, a mustard poultice externally, and sucking ice at frequent intervals may be ordered. A pastil containing morphine (gr. $\frac{1}{2}$), cubebs (gr. $\frac{1}{2}$), atropine (gr. $\frac{1}{16}$), proto-iodide of

mercury (gr. $\frac{1}{60}$), may be given every four hours, till six have been taken. If only a few hours can be given for treatment, a hypodermic injection of gr. $\frac{1}{30}$ of strychnine may be given. Twenty minutes before the engagement a glass of vin Mariani de coca should be taken, and the following solution be used with an atomiser and well inhaled:—

R	Menthol	-	-	-	-	-	grs. vj
	Morphine	-	-	-	-	-	gr. ss
	Cocaine	-	-	-	-	-	grs. ij
	Colourless vaseline oil	-	-	-	-	-	℥ss

In young children there is almost invariably some febrile disturbance, and minute doses of aconite frequently repeated should be administered to reduce the temperature. Confinement to a warm, properly ventilated room is obviously essential; and if there is considerable respiratory embarrassment, the patient should be placed in a steam bed, and hot moist applications kept on the throat. Ipecacuanha, apomorphine, or tartar emetic should be given frequently in small doses. When there is difficulty in coughing up the viscid expectoration, the early administration of a non-depressing emetic, such as ipecacuanha wine or sulphate of zinc, often affords marked relief. Free evacuation of the bowels is important in children as in adults.

In spasmodic laryngitis minute doses of trinitrine frequently repeated (gr. $\frac{1}{1000}$ to gr. $\frac{1}{500}$) every two or three hours will often relieve the spasm; or caffeine, bromide of potassium, or iodide of sodium may be tried with advantage.

If laryngeal obstruction persists, it may be necessary to perform intubation, or tracheotomy. To my mind there is no doubt that intubation should be preferred if medical aid can be summoned within half an hour, should the tube be coughed up. The great danger, next to acute asphyxia, is the occurrence of wide-spread pulmonary engorgement and catarrhal pneumonia, and, since this is certain to come on should the urgent dyspnoea be allowed to continue, it is better to intubate quite early, for in non-membranous laryngitis, and in skilled hands, the procedure is practically unattended with danger. (See Intubation, p. 213.)

After recovery from the acute symptoms great care must be exercised in preventing a recurrence of the attacks, and exercise in the fresh air, plain food and warm clothing, will be necessary.

MEMBRANOUS CROUP.

Membranous laryngitis is generally laryngeal diphtheria, but not always, and it appears probable that non-diphtheritic membranous croup occurs with greater frequency than is generally believed. Cases of membranous laryngitis may therefore be divided into two groups: (1,) Membranous croup; and (2,) laryngeal diphtheria, which is described in the chapter on diphtheria (see page 134).

Etiology and Pathology.—While there is good reason to regard idiopathic membranous croup as a definite and distinct affection, it must be remembered that caustics or irritants, such as strong ammonia, chromic acid, scalds, the galvano-cautery, will cause a membranous exudation indistinguishable from the membrane of the idiopathic affection. A membranous laryngitis has also been observed in cases of scarlet fever, small-pox, and typhoid fever, and the occurrence of a *membranous* exudation in these affections is due to streptococcic infection.

There is no longer room for doubt as to the occurrence of idiopathic membranous croup distinct from diphtheria. It frequently happens that in true diphtheritic croup bacteriological examination gives negative results as regards the Klebs-Loeffler bacillus, owing to the culture being imperfectly inoculated, but many cases have been observed in which the examination of the membrane was made by competent observers on several occasions by swabs and cultures, and in which the Klebs-Loeffler bacillus was invariably absent. A series of eight cases in point occurring at the Bristol Royal Infirmary and Children's Hospital have been recorded by Symes, and similar instances have been reported by Gougenheim, Glover, and others.



FIG. 70.

Bronchial cast expectorated by a child at the Bristol Royal Infirmary under Dr. Shaw. Vincent's spirillum was found by Dr. Symes on bacteriological examination, but Klebs-Loeffler bacilli were not present. (Quarter actual size.)

Membranous croup generally attacks children between the ages of two and eight, and it is most commonly caused by exposure to damp and chilly atmospheric conditions.

Symptoms.—Membranous croup is generally primary in the larynx, but the false membrane may originate in the fauces or pharynx, spreading thence to the larynx. It may begin with the usual symptoms of a catarrhal laryngitis, but in the course of an hour or two, if not more suddenly, a characteristic loud brassy cough comes on, and gets worse especially towards midnight. The child becomes restless, and the temperature rapidly rises till it reaches 102° or 103°. The cough, at first



FIG. 71.

Membranous laryngitis in a boy aged 18. Cover-slip stained with gentian-violet showed numerous bacilli, but a streak culture on Kanthack medium yielded no growth. Agar culture showed only the presence of streptococci with a few staphylococci. Course very mild.

occasional only, becomes more frequent, and the attacks are often followed by laryngeal spasm. In the space of a few hours the symptoms of laryngeal obstruction appear, and the child endeavours to cough out the obstructing matter, clutches at its throat, and is more restless. Before long the cough is characteristically croupy, and the respiration becomes more and more embarrassed as the membrane is formed in the larynx, till, owing to its presence, the voice

and cough become aphonic (silent croup), and the air forced through the narrowed glottis produces the peculiar crowing sound.

It is not unusual for some remission of the symptoms to occur towards morning, so that the acute stage may not be reached till the evening of the second or third day, but the course of membranous croup differs from the catarrhal form in its being *progressive*. When the acuter stage has been reached the cough may be frequent, and possibly may result in getting rid of some of the obstructing membrane, with temporary relief to the breathing. When the glottic obstruction is well marked there is great inspiratory and expiratory dyspnœa, and asphyxia may be threatened, necessitating intubation or tracheotomy.

At first the pulse is hard and frequent, and the affection pursues a sthenic course; but if the obstructive dyspnœa continues, the deficient supply of oxygen and the carbonic acid

toxæmia cause a fall of temperature and rapidly progressive weakness which make the case very difficult to differentiate from diphtheria. If the membrane be thick or extensive fluids may regurgitate through the nose on drinking, owing to the mechanical interference with the movements of the soft palate, but paralytic sequelæ do not follow the attacks as in diphtheria.

The foregoing description applies to cases arising in children. But in adolescents and adults the symptoms may be of a mild character throughout, resembling acute laryngitis. St.Clair Thompson has observed a blue membranous laryngitis due to the bacillus pyocyaneus.

The **Prognosis** is often very grave; it may terminate fatally in twenty-four hours, or may last seven or eight days. Hilton Fagge gave the mortality at 60 to 70 per cent., while Morell Mackenzie put the recoveries of cases untreated by tracheotomy as low as 10 per cent. Of five hundred and five cases of true croup, collected by McNaughton and Maddren, and treated by calomel fumigations, intubation, or tracheotomy, 54·5 per cent. recovered; while of two hundred and seventy-five cases treated by calomel fumigations alone, 48·7 per cent. recovered. It is probable that some of the above were really cases of diphtheria, but there is no room to doubt the gravity of the disease. Of Symes' eight cases half the number died.

In adults, however, a membranous laryngitis may be very mild in its course and devoid of dangerous symptoms.

Diagnosis.—Membranous croup requires to be differentiated from spasmodic croup, laryngismus stridulus, diphtheria and retro-pharyngeal abscess.

The differentiation between non-bacillary croup and diphtheria cannot be made with certainty without a bacteriological examination, but speaking generally the onset of croup is much more rapid than that of true diphtheria, and the temperature rises more quickly, reaches a higher point, and is more persistent. The membrane has not the dead white or whitish-grey appearance of diphtheritic membrane, but is generally an entirely superficial deposit, friable, soft, and yellowish in colour, is easily detached, and does not leave an ulcerated or bleeding surface.

Treatment.—At any rate until repeated bacteriological examination has proved the case not to be diphtheria, the

difficulty of altogether satisfying oneself that any case is simple membranous laryngitis, renders it advisable to treat every case as though it was diphtheria, and all measures that tend to exhaust the patient should be studiously avoided.

At the outset it is well to administer a non-depressing emetic, such as ipecacuanha wine or powder. The patient should be placed in a steam bed, and hot fomentations applied to the throat, for such measures aid in the expulsion of the membrane. My usual practice is to administer calomel in doses of one to two grains, according to the age of the child, repeated every two or three hours till the bowels have acted freely, and then at less frequent intervals. The administration of calomel in membranous croup was advocated by Dundas Grant many years ago. When the mercurial salt, having acted on the bowels, is being given at longer intervals, perchloride of iron should be prescribed. Spraying the larynx can only be adopted in older children. Peroxide of hydrogen spray is praised by Glasgow: "The liberation of gas which takes place seems to raise the membrane from its attachment, and so facilitates its expulsion." I have no personal experience of its use in membranous croup.

Corbin's method of treatment by calomel fumigations has yielded excellent results in the practice of many American physicians. A dose of one or two grains of calomel is given before the sublimations, which should be begun early, as soon as the diagnosis of true croup can be made. The patient is placed in a completely enclosed steam tent bed, and the calomel takes about ten minutes to volatilise, the tent bed being kept closed for fifteen minutes. "A very safe method is to volatilise grs. xv every two hours for two days and nights, then prolonging the intervals to three hours on the third day, four hours on the fourth day, fumigating three times daily thereafter according to indications" (O'Dwyer). If pure calomel is used, ptialism rarely occurs, but anæmia is prone to occur, and should be combated by the administration of iron.

CHRONIC LARYNGITIS.

Etiology.—Chronic laryngitis in a large percentage of cases dates from an acute or subacute attack, and thus all causes of acute laryngitis are important etiological factors. It may be chronic from the outset, and due to excessive or faulty use of

the voice, working in ill-ventilated rooms, or breathing in dusty atmosphere, or to chronic dyspepsia, and the abuse of alcohol or tobacco. It is often associated with chronic pharyngitis and rhino-pharyngitis, or general anæmia. Persistent laryngitis often precedes tuberculosis of the larynx or lung; it is also predisposed to, or directly caused by, the rheumatic or gouty habit. Undoubtedly a prolific cause of intractable laryngitis lies in nasal obstructions, with resulting mouth-breathing.

Symptoms.—The chief symptoms are huskiness of the voice in varying degree, and vocal weakness. The patients complain that the voice is quickly tired, and becomes hoarse, or goes altogether; there is a sense of dryness or tickling in the larynx, and, in the hyperplastic forms, slight or considerable obstructive dyspnoea. Cough is often complained of, with little expectoration, unless, as is so often the case, the trachea and bronchi are likewise the subjects of chronic catarrh.

Examination shows in simpler cases that the laryngeal mucous membrane is somewhat redder than usual, either diffusely or in patches. The vocal cords are slightly pink or grey, and sometimes small vessels may be seen coursing over them. Small accumulations of mucus may be found on the ventricular bands and vocal cords, or lying along their free margins, the sticky



FIG. 72.

Strings of sticky mucus stretching across the glottis in laryngorrhœa.



FIG. 73.

Pachydermia diffusa of the vocal cords, and of the inter-arytenoid fold.

mucus stretching across the glottic opening, especially near the anterior commissure. When persistent and excessive, the term *laryngorrhœa* is sometimes applied to these cases. *Fig. 72* is taken from a professional bass. He dated all his trouble from straining the voice in using the “coup de glotte” in shouting to a friend.

Pachydermia laryngis, etc.—Other clinical varieties which occur have been termed “glandular laryngitis,” in which not only is the mucous membrane thickened, but there is also enlargement of the racemose glands; again, there is the hyperplastic form, *hypertrophic laryngitis* or *pachydermia diffusa* (see Plate XVI, Figs. 5 and 6). Rice describes a condition of “choked voice” in which the ventricular bands become thickened and overlap the true cords, whose function they seem to replace in some measure. He attributes it to faulty method in using the voice by those who speak or sing in excess. If the hyperplasia be localised, the most common situation of the hypertrophy is in the posterior commissure, causing wrinkling of the mucous membrane, or mammilliform outgrowths, which may interfere mechanically with the approximation of the cords, or the vocal cords may show nodules on the vocal processes (*pachydermia laryngis*, Virchow). The ventricular bands are often thickened so considerably as to conceal the vocal cords altogether. A form analogous to chronic follicular pharyngitis is termed *granular laryngitis*.

In a condition described by Störk as *blennorrhœa* of the nose, larynx and trachea, a subglottic infiltration arises which is said to result frequently in adhesion of the anterior portions of the vocal cords. This was regarded by Gangholmer as a subglottic laryngitis, and Schrötter believed that under the term blennorrhœa Störk had included two varieties of cases, *viz.*, *ozæna laryngis* and a form of rhinoscleroma. Störk's cases may perhaps be most correctly described as chronic subglottic laryngitis due to rhinoscleroma.

The subglottic form of hyperplasia, *chorditis vocalis hypertrophica inferior*, shows the infra-glottic swelling below the vocal cords on deep inspiration.

The *œdematous* form is characterised by diffuse or localised œdematous infiltration, while the *hæmorrhagic* variety is attended with marked hyperæmia followed by hæmorrhage.

In another variety, *laryngitis sicca*, the secretion is scanty, the patient occasionally coughing up dry crusts of inspissated mucus which may be streaked with blood. At times, too, the collection of these dry crusts below the glottis may cause intermittent attacks of dyspnœa, or temporary aphonia may be caused by their adhering to the cords.

Superficial catarrhal ulceration, especially of the vocal cords, may occur, but it should lead to the suspicion of tuberculosis.

Submucous laryngeal hæmorrhage.—A variety of causes may

Inflammatory Affections of the Larynx.

FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.



FIG. 5.



FIG. 6.

FIG. 1.—Catarrhal laryngitis, with hyperaemia of the cords and paresis of the internal tensors.

FIG. 2.—Inflammatory oedema of the larynx. The epiglottis and aryteno-epiglottidean folds are enormously swollen, concealing the glottis.

FIG. 3.—Abscess resulting from perichondritis of the right ala of the thyroid cartilage, causing inflammatory swelling of the right ventricular band; ary-epiglottic fold and subglottic oedema. (Greville Macdonald.)

FIG. 4.—Oedema of the right ary-epiglottic fold, and to less extent on the left, and subglottic oedema on the right side.

FIG. 5.—Pachydermia laryngis in the interarytenoid space.

FIG. 6.—Pachydermia laryngis of the vocal cords.

give rise to superficial laryngeal hæmorrhage with hæmoptysis (see p. 87), but the hæmorrhage from these same causes may be submucous. A submucous laryngeal hæmorrhage, especially if into the vocal cords, may give rise to symptoms of chronic laryngitis. But laryngoscopic examination will reveal the bright red or purplish effusion of blood beneath the mucous membrane of the affected area. Semon records three cases in which sub-mucous blood-clots simulated neoplasms of the larynx, viz., an angioma, cancer of the vocal cord and soft fibroma, causing much difficulty in diagnosis.

Treatment.—Any constitutional condition that may act as a predisposing cause of the affection, *e.g.*, rheumatism, gout, dyspepsia, anæmia and constipation, must of course receive attention; and whatever is the directly exciting cause, any departure from a condition of perfect health, must be remedied if we hope to afford lasting relief from such an intractable affection as chronic laryngitis. Any evidence of nasal obstruction should be noted. The voice should be used as little as possible, and all exciting causes, whether lying in the habits or occupation of the patient, should be avoided.

In clergymen, schoolmasters, vocalists, and public speakers, laryngitis is very frequently brought on, not so much by excessive use of the voice as by wrong vocal methods, and the importance of attention to this point cannot of course be over-estimated. Ellis has shown that in schoolmasters the inhalation of chalk dust from the blackboard is a frequent cause of the affection.

As regards the loss of the singing voice, the vocal failure is often due to either: (1,) wrong methods of breathing; (2,) the wrong use of the vocal registers; (3,) the simple over-use of the voice; or (4,) the use of the voice while suffering from catarrh (Hunt).

I have already alluded to the importance of attending to the general health, and a simple tonic treatment, such as the administration of iron and quinine, strychnine and phosphorus will often prove highly beneficial, while change of air to a dry bracing climate, fresh air and exercise, a morning cold bath and similar measures, are as important as the administration of drugs.

Of internal "specific" remedies the most useful in my experience are iodide of potassium or the proto-iodide of mercury

(gr. $\frac{1}{16}$) three or four times daily, and cubebs or ammonium chloride. Muscular tone will often be improved by strychnia in full doses. Locally, stimulating inhalations of turpentine, oil of Scotch pine, camphor, creasote, or eucalyptus, may be used at night; and during the day the oil atomiser of eucalyptol (gr. xx), camphor (gr. j to ij) to the \mathfrak{z} j of liquid vaseline, or inhalations of nascent chloride of ammonium vapour, are beneficial.

Pastils containing benzoic acid, or chloride of ammonium, or cubebs (gr. $\frac{1}{2}$) and guaiacum resin (gr. ij), with emetin (gr. $\frac{1}{32}$) may be very useful, three or four being dissolved in the mouth daily.

As a rule applications to the larynx should be avoided, unless there are hypertrophic changes. Then such astringents as

chloride of
zinc (grs. x

to xxx to

FIG. 74 Laryngeal Brush ($\frac{1}{2}$ size showing the proper form and size.

\mathfrak{z} j), or alum (grs. v to x, or perchloride of iron (grs. ij to

xx), may be applied with the laryngeal brush or cotton

wool holder, or what is far pleasanter to the patient, a

laryngeal spray. In making these applications the

laryngoscopic mirror is held in the left hand, and thus

the brush can be guided by sight, the patient himself holding

the tongue in a small towel. When a laryngeal spray or dropper

is used, the application must be cautiously made only during

phonation.

If the voice remains weak after the inflammation and congestion have subsided, the local application of the faradic or galvanic current will sometimes effect an improvement.

When chronic tracheitis and bronchitis are associated with the laryngeal affection, inhalations and the topical application of menthol in olive oil, or creasote, guaiacol (2 per cent.), salol (20 per cent. to 30 per cent.) in olive-oil by intra-laryngeal injections, have yielded excellent results in the hands of Grainger Stewart, Bronner, Sharp and Downie. About \mathfrak{z} j of the oily solution is injected daily straight into the trachea by means of a special syringe, preferably Bronner's silver and glass syringe. Care must be observed to pass the nozzle of the syringe well below the glottis during a deep inspiration, before injecting the contents. Obstinate cases often improve by change of air, a sea voyage or a course of waters at some spa, such as Ems, Carlsbad, or Aix-le-Bains.

In the treatment of *pachydermia laryngis* there is great diversity of opinion. If the use of alcohol or tobacco has been excessive, the habit must be corrected; if the laryngeal condition is associated with chronic pharyngeal catarrh or laryngitis, treatment appropriate to these conditions must be pursued. Rest and mild astringent laryngeal sprays will produce a favourable result in slight cases. Internally proto-iodide of mercury in gr. $\frac{1}{10}$ doses, three times daily, has appeared to me to materially lessen the deposit. The administration of small doses of iodide of potassium has many advocates. Locally, the repeated application of alcoholic solutions of salicylic acid (1 to 6 per cent.) will sometimes reduce the thickenings. Scheinmann has obtained good results from the use of steam inhalations of a 2 or 3 per cent. solution of acetic acid for ten minutes, three times daily, for some weeks. O. Chiari, to prevent recurrence, recommends electrolysis, as employed by Moll, viz., a current of ten to twelve milliampères for from three to five minutes at a time.

Small submucous hæmorrhages will generally be absorbed spontaneously; the larger effusions may be liberated by incision and turning out the clot.

CHORDITIS TUBEROSA.

Chorditis Tuberosa, or "singer's nodule," is a clinical variety of pachydermia. A peculiar, small, pale poppy-seed-like growth appears on the upper surface and free border of a vocal cord, about the junction of the anterior third with the posterior two-thirds, and is surrounded by a zone of hyperæmia. On the opposite cord there is often a corresponding depression. These nodules are the result of improper voice production, and are most commonly seen in sopranos. These wrong methods arise especially in the production of very high notes in the small register, the posterior two-thirds of the cords being held in apposition and only a small chink left anteriorly; hence the reason for this particular nodal point being subject to constant attrition in sopranos. It is remarkable how comparatively little the voice may be affected; indeed, I once had a patient who, although he

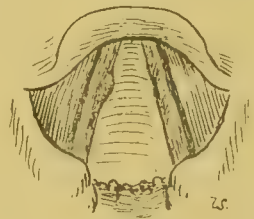


FIG. 75.
Chorditis Tuberosa.

was a distinguished tenor, had continued to sing up to the time he came to me, in spite of the nodule and hyperæmia of the cords shown in the figure. After a period of absolute rest, and the constant application of thuja and other local treatment, he completely recovered, and only then realised how much his voice had been affected. His chief difficulty was in passing smoothly from the upper to the middle register, and the lower notes were impaired most.

Treatment.—This requires considerable patience and perseverance, if any permanent benefit is to result. The first essential is rest. When congestion has subsided the nodule may be removed by a sharp curette, and a mild local astringent solution, *e.g.*, chloride of zinc, 20 grs. to the oz., applied daily for a week. Mackenzie advised simply the application of perchloride of iron solution. Rice advises removal by a small snap-shot guillotine and chloride of zinc; while Bosworth prefers nitrate of silver (30–60 grs. to the fl. oz.), and avoids the use of forceps. I rely chiefly on rest and small doses of iodide of potassium and the proto-iodide of mercury, as in chronic laryngitis, and the correction of any faulty method in singing. Great care must be exercised in removing these nodules, and if this be too successfully done before the effect of prolonged rest has been tried, it is possible that in the course of time an indentation of the margin of the cord will mark the position of the former nodule.

Too little attention is often paid to the all-important question of improper vocal methods as a cause of these nodules in singers. Holbrook Curtis claims that he is able to restore the voice and cause the disappearance of singers' nodules, without the aid of local applications and by means of his vocal and respiratory exercises alone. Though my personal experience of his methods is too limited to confirm all that he claims, I have found these exercises of very great value and productive of gratifying results. Briefly, Curtis insists on the lower costal respiration with the upper ribs elevated; while as regards the vocal exercises "the object, primarily, is to make the cords adopt a new method of vibration in respect to their segmentation, and this is accomplished by changing the colour or overtone effects. To do this, we must cover the tone and make the initial sounds seem to arise from the resonators of the face. The sound made with the mouth closed and commonly written 'humph' illustrates this as

well as it can be described in print. Now, in the place which we call the focus of this sound we should start the purest and most musical note we are able, imitating the sound of a distant steam whistle, preferably on *c''* of the staff for a soprano and *c'* for a tenor. This having been accomplished, we strive by a mental effort to bring this tone to the lips, the mental tone picture being transformed into the word *ma*, as in mah or maw, and with this labial tone thought dominant the lips should be separated by dropping the lower jaw, the lips not sharing in the least in the muscular effort. If this tone has also taken possession of the buccal cavity—in other words, if the mouth is made a resonator, and the tone is sufficiently far forward—the musical note or hum will be greatly accentuated by opening the mouth in this manner, and the sound will appear to have its origin upon the lips."

For fuller information on the exercises and their practical application the reader should refer to Curtis' work, "Voice Building and Tone Placing."

CONGENITAL LARYNGEAL STRIDOR.

It is convenient to consider here an affection characterised by a peculiar inspiratory stridor, commencing at or soon after birth, which has been variously designated "Respiratory croaking in babies" (Gee), "Infantile laryngeal spasm" (Goodhart), "Infantile respiratory spasm" (John Thomson).

Etiology and Pathology.—The symptoms are probably due to congenital laryngeal malformation, and not associated with true glottic spasm; the term "congenital laryngeal stridor" therefore sufficiently describes the condition.

But the complaint has also been regarded by various observers as the result of (*a*), a spasmodic affection of the glottis; (*b*), compression and consequent stenosis of the trachea by an enlarged thymus; and (*c*), paralysis of the postici muscles, etc. The persistence of the stridor makes it very difficult to accept any view implying such a long continued laryngeal spasm: while in some cases there has been no enlarged thymus, and in others in which an enlarged thymus existed there was no stridor or, if present, it was expiratory as well as inspiratory.

Sutherland and Lack, as the result of direct laryngoscopic examination in a number of cases, are convinced that the stridor

depends on a valvular action of the upper aperture of the larynx, a falling inwards of its lateral walls during inspiration, dependent partly on a peculiar congenital malformation of the larynx, and partly on the flaccidity of these parts in infants. Thompson and Logan Turner have observed a similar conformation of the larynx, and have reproduced it artificially in the cadaveric larynx (of children) by imitating forcible inspirations through the glottis. Thompson, however, considers that in life the laryngeal deformity arises from disturbances of respiratory co-ordination resulting in a constant sucking in of the upper margins of the infantile larynx, "a process strictly analogous to the formation of pigeon-breast:" while Sutherland and Lack, on the other hand, believe that the peculiar congenital formation of the larynx is the essential cause. Probably both factors co-operate; thus, respiratory disturbances arising in an infant presenting the special infantile type of larynx described above causes stridulus inspiration, and the falling in of the soft yielding tissues increases the inspiratory stridor and results in a persistently pronounced laryngeal deformity.

Symptoms. The stridor commences at or soon after birth, and though its onset may be delayed until the end of the second week, it rarely develops after that time; but a case terminating fatally soon after coming under treatment, at the age of twenty-two months is recorded by Variot. The little patients may appear in good health apart from the laryngeal condition, though often they are subject to bronchitis, and are rickety or otherwise unhealthy. Thomson's statement that "inspiration begins with a croaking noise and ends in a high-pitched note; expiration is accompanied by a short croak when the stridor is loud, but at other times is noiseless," is descriptive of a typical case, but the inspiratory stridor may be fairly "a deep note, purring or growling, or high-pitched, clear, or squeaking, and the expiratory sound is sometimes of a nasal or pharyngeal character" (Sutherland and Lack). The inspiratory stridor is always very much louder than the expiratory; the loudness of the stridor varies at times; and there are generally longer or shorter periods of complete intermission. When the child is restful and breathing is quiet, especially when asleep, the stridor is least marked; but if it cries, coughs, gets excited, or is exposed to cold, or from any cause respiration is hurried, the stridor is increased.

The usual physical signs of obstruction to respiration are present during the exacerbations, such as cyanosis, retraction at the epigastrium and lower ribs, supraclavicular fossæ, and episternal notch. As a rule the lungs are not fully expanded, breath sounds being deficient at the base. When the stridor was pronounced and had lasted for some months, Sutherland and Lack found that the boundaries of the chest became altered, as in rickets. Further, on laryngoscopic examination, they found in all of their cases the following characteristic appearances: The epiglottis was sharply folded on itself, the two lateral folds being in close apposition, and in some cases in contact. The aryteno-epiglottic folds were approximated, and thus the upper aperture of the larynx was reduced to a long narrow slit. The thin folds bounding this aperture seemed quite flaccid, and flapped to and fro on respiration. The inspiratory column of air striking down on these folds drove them together, and on expiration they again separated. In some of their cases, the "purring" ones, the coarse vibrations of these folds could be distinctly seen.

As the child grows the malformation described persists in some measure for years, but the stridor passes off as the parts forming the superior laryngeal aperture become less yielding. Sutherland and Lack point out that the fact that the folds are wider apart at the commencement of inspiration, and close during its course, agrees with the higher pitch of the stridor at the end of inspiration, when the chink is narrowest, while the flapping outwards also accounts for the diminution or absence of the stridor on expiration.

Lack never observed the malformation except in association with congenital stridor, though he systematically examined the larynx of every infant coming under his care for many months, and in no case did he find much enlarged tonsils or adenoid vegetations.

Diagnosis.—The conditions liable to be mistaken for congenital laryngeal stridor are laryngismus stridulus and other forms of glottic spasm, papillomata, laryngitis and spasmodic croup, etc. The early onset of the more or less persistent peculiar stridor, with signs of obstructed respiration, and with clear vocal utterance, is fairly characteristic, while laryngoscopic inspection by Lack's method (see p. 49) reveals the cause of the condition. Attention to these points serves to differentiate the affection.

Prognosis.—The majority of cases outgrow the affection by the end of the second year, or even before then, but a fatal termination is by no means rare. If the health remains good and the respiratory embarrassment is moderate, the prognosis is favourable; but the existence of a rachitic diathesis, bronchitis, or any severe attack of illness, is liable to be attended with increased dyspnœa, which may rapidly terminate in acute asphyxia.

Treatment.—It is important to ensure good general nutrition, and to avoid exposing the patient to cold or any conditions which may induce catarrhal affections of the respiratory tract. Weakly children should have cod-liver oil, malt extract, and the diet should be carefully regulated, while any manifestation of rickets or bronchitis should receive immediate attention. When respiration is much embarrassed, tracheotomy should be performed, and in the very young it is most desirable not to delay this so long that the lungs become congested and engorged with blood beyond the possibility of recovery.

ŒDEMA OF THE LARYNX.

Two varieties of œdema of the larynx may be described, viz., inflammatory œdema, primary or secondary, and non-inflammatory or passive œdema.

ACUTE INFLAMMATORY ŒDEMA.

Simple or non-infective œdema may be due to injury from swallowing some hard or pointed body, or from some strong irritant inhaled, swallowed, or applied to the larynx.

Etiology.—Inflammatory œdema (*see Plate XVI, Fig. 2*) supervenes rarely on an acute catarrhal laryngitis. It is sometimes more or less localised as an *epiglottitis* (miasmatic), *arytenoiditis*, or *chorditis*. *Laryngitis hypoglottica* may be described as an acute subglottic œdema. Inflammatory œdema may occur in scarlet fever, typhoid fever, and small-pox; and Pugin Thornton has seen three cases following influenza. Acute infectious phlegmon of the larynx may arise by an extension of acute phlegmonous pharyngitis, or this very fatal disease may be primary in the larynx (*see p. 74*); and most of the cases of so-called idiopathic œdema or abscess of the larynx are probably of this nature; acute primary œdema of the larynx from other

causes being very rare. According to Sestier's statistics on œdema of the larynx, simple inflammation was the cause of œdema in only 6 per cent. of all his cases.

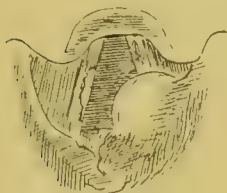


FIG. 76.
Perichondritis and secondary œdema of the left arytenoid and ary-epiglottic fold in a tubercular larynx.

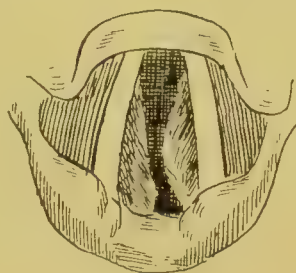


FIG. 77.
Laryngoscopic appearance in subglottic œdema.

Acute secondary œdema may occur in syphilitic, cancerous, or tubercular (rare) disease of the larynx.

Symptoms.—Excluding the septic cases, which are considered elsewhere (see p. 74), the symptoms are those of catarrhal laryngitis, with more pain, dyspnœa, and greater constitutional disturbances super-added. When the epiglottis is implicated, there is difficulty in deglutition; and, if the arytenoid folds and ventricular bands are involved, respiration will be embarrassed from the resulting laryngeal stenosis, more especially if the sub-glottic tissues are implicated.

Objectively the epiglottis is seen as a pink sausage-shaped swelling, and beneath it the swollen rounded arytenoids and ary-epiglottic folds, which lose their normal contour, and conceal the infiltrated ventricular bands and the subjacent vocal cords. When the parts above the vocal cords are not much affected, it may be possible to observe subglottic swelling.

Treatment.—*Acute inflammatory œdema* should be treated on the same lines as the severer forms of acute catarrhal laryngitis; but in the more urgent cases leeches should be applied to the larynx externally, followed by the ice-bag, and free scarification of the œdematous swelling should be always performed, if practicable. The swelling then generally rapidly subsides, and though it may recur, and the scarification have to be repeated, the relief is usually very marked. Should it fail to relieve the threatening asphyxia, tracheotomy should not be delayed. The trachea should be opened low down, as subglottic œdema is

generally associated with the supraglottic inflammation. Intubation may be tried. In one case I was successful in relieving the patient, an adult, by this means, when almost suffocated, and though the tube had to be replaced once or twice, as it was only a child's tube, the necessity for tracheotomy was obviated. As a matter of fact, the intubation instruments were at hand, and there was no time to get tracheotomy instruments in this case ("Brit. Med. Journ.," 1888, i, p. 745).

Scarification may be done in some cases with the aid of a laryngeal mirror. Mackenzie's or Heryng's scarifier may be used; or, failing any special instrument, an ordinary sharp-pointed curved bistoury, with all but the last half-inch of the blade wrapped in lint or in plaster. If dyspnœa is urgent, the larynx may be scarified without inspection, guiding the punctures by the left forefinger tip in the larynx.

NON-INFLAMMATORY ŒDEMA.

Etiology.—It occurs in renal disease with or without general anasarca, and very rarely in diabetes and obstructive valvular heart disease. It may be produced by local injury, or by pressure on the veins of the neck by tumours, or from the internal administration of iodide of potassium. It sometimes supervenes in syphilitic or cancerous affections of the larynx, and, more rarely, in tubercular disease and diphtheria. Strübing describes a form of œdema which he calls angio-neurotic, which appears and disappears very rapidly, and is generally associated with a similar condition occurring in the face.

Symptoms.—In non-inflammatory œdema the symptoms are simply those due to laryngeal obstruction, or to mechanical interference with the action of the vocal cords, and vary with the extent and locality of the inflammation. Objectively the parts affected are swollen and translucent, the contour of the laryngeal structures being much the same as in inflammatory œdema (see *Plate XVI, Fig. 4*).

Treatment.—*Non-inflammatory œdema* may call for relief by scarifying, or require tracheotomy; while appropriate general treatment is indicated in renal and cardiac affections, and in syphilis, etc. Pilocarpine, injected hypodermically, has been used with success.

CHONDRITIS AND PERICHONDRITIS OF THE LARYNX.

Fibroid degeneration of the cartilages has been described, but is an extremely rare condition, and one I have never met with.

Ossification of the laryngeal cartilages generally occurs more or less extensively as old age approaches, and is simply one of the indications of the degenerative changes in the tissues of those advanced in years. It sometimes takes place in middle life but is without significance.

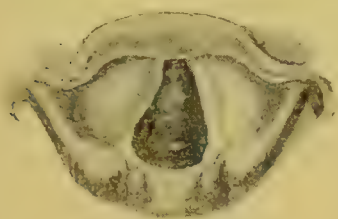


FIG. 78.

Syphilitic perichondritis of the right ala of the thyroid cartilage, giving rise to a swelling beneath the right ventricular band.

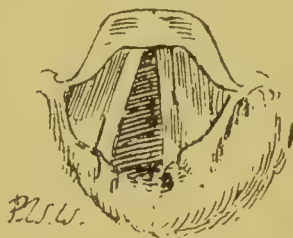


FIG. 79.

Perichondritis of the cricoid cartilage.

Etiology and Pathology.—Primary perichondritis is decidedly rare and usually associated with acute or chronic catarrhal laryngitis, due to cold. The great majority of cases are secondary, although in many cases the immediate cause may be difficult to determine. Acute perichondritis is most frequently due to syphilis, tubercle or malignant disease, typhoid fever, variola, diphtheria, and other infectious fevers, and may arise from septic laryngitis, gout, injury by swallowing hard or pointed matters in food, or the frequent or careless passage of œsophageal bougies. If the perichondrium of the inner surface is the seat of the inflammation, an irregular, nodular, unilateral, inflammatory swelling can generally be recognised, encroaching on the subglottic space, and pushing up the vocal cord; or the posterior surface only may be affected.

The thyroid perichondrium may be inflamed internally, forming a smooth red swelling beneath the ventricular band. If the external surface is the seat of the inflammation, tenderness and swelling over the thyroid will be obvious.

The arytenoid is sometimes the only cartilage affected, appearing as a red smooth swelling, involving more or less fixation of the corresponding cord.

Apart from syphilis, tubercle, and malignant disease, the cricoid cartilage is the one most frequently attacked. Though the arytenoid cartilage is generally involved, perichondritis may be limited to the cricoid cartilage.

Symptoms.—Acute perichondritis is often ushered in with chilliness or a rigor; the temperature in such cases is febrile. When the cricoid is involved on its inner surface, considerable dyspnœa and aphonia may result from the subglottic swelling. If the perichondritis is on the posterior surface, pain on deglutition is a prominent feature.

The exudation and swelling may undergo resolution, though, even in the milder cases, there is a tendency for fibroid degeneration of the products of inflammation, resulting in what Semon has called "adhesive perichondritis" and ankylosis of the crico-arytenoid joints. But in many cases, owing to the scanty vascular supply of the perichondrium and the absence of vessels in the cartilage itself, the separation of the perichondrium from the underlying cartilage very often results in suppuration, with necrosis of the affected cartilage. This is especially liable to follow cricoid or arytenoid perichondritis. In these cases of suppurative perichondritis, purulent exudation may persist for months or years until the necrosed sequestrum is exfoliated, during which process the patient presents a miserable aspect, and becomes greatly emaciated from the pain and dysphagia. Ultimately cicatrisation generally occurs, with marked laryngeal deformity, sometimes causing considerable constriction and dyspnœa.

Diagnosis.—The tumefaction due to perichondritis may be mistaken for tubercular disease or syphilis, and in the latter affection it may be impossible to determine whether one has to deal with a tertiary deposit or perichondritis until necrosis has occurred, except from the history and the result of antisymphilitic remedies. Tubercular disease is rarely attended with suppuration, and the general condition of the patient will throw light on the case.

The painful swelling, with deep, angry-looking ulceration, may closely resemble malignant disease, and in doubtful cases it may be necessary to remove a portion of the swelling for microscopical examination.

Prognosis.—The milder cases are prone to leave their traces in

PLATE XVII.



Fig. 1.

Necrosis of the Cricoid Cartilage. The perichondritis involving the posterior three-fourths of the cricoid cartilage resulted in suppuration around the cartilage. The abscess opened into the larynx near the posterior extremity of one vocal cord. There appears also to have been another smaller abscess at the right side of the epiglottis.

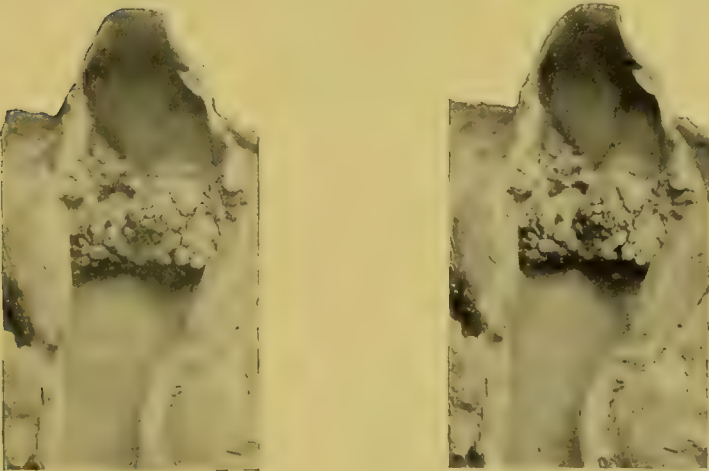


Fig. 2.

Papilloma laryngis, showing an exceptionally large amount of the cauliflower excrescences on both sides.

the formation of cicatricial contraction or crico-arytenoid fixation, but the occurrence of suppuration and necrosis of cartilage is followed by a long period of discharge with septic fever, and the patient may succumb during the process.

Treatment consists in the application of ice externally, giving the patient ice to suck, and treating the general symptoms. If dyspnœa is marked, laryngotomy may be required. When suppuration has occurred, with consequent necrosis, the dangers are considerably increased, and many patients succumb to this disease; therefore the strength must be supported by general tonics and dietetic measures. If possible the necrosed sequestrum should be removed, as early as it can be accomplished.

DISEASES OF THE CRICO-ARYTENOID JOINT.

INFLAMMATION, ANKYLOSIS, AND LUXATION.

Definition.—Any degree of stiffness of the crico-arytenoid joint is included under the term “ankylosis,” whether it be due to disease of the joint itself (“true ankylosis”) or to disease in the immediate neighbourhood of the joint which mechanically interferes with the movements of the arytenoid cartilage (“false ankylosis”).

Luxation consists in partial or complete displacement of the arytenoid cartilage from its articular surface on the cricoid. Luxation and ankylosis of the arytenoid cartilage may co-exist.

Etiology.—Of course, all acute inflammatory affections involving the arytenoid cartilage, or the mucous membrane covering it, are very likely to involve the crico-arytenoid joint, or at least to cause fixation of the arytenoid cartilage from inflammatory exudation. Such conditions are prone to arise in perichondritis, gouty and rheumatic attacks, the exanthemata, diphtheria, and from mechanical injury, while cancer, syphilis, and tubercle may be cited as frequent causes of less acute ankylosis, with or without arthritis.

The **Symptoms** of inflammation involving the crico-arytenoid joint vary according to the nature of the affection causing it, and they may be simply those of laryngitis. But if the tumefaction is considerable, the resulting ankylosis causes fixation of the vocal cord. The vocal cord may in this way be fixed in any position, and if it be in the median line the voice may be scarcely altered at all. More often it assumes the cadaveric

position, and rarely the position of abduction; in such cases the voice will be impaired or altogether lost. If both sides are involved, the vocal cords may not be bilaterally symmetrical in position; but dyspnoea or rapidly fatal asphyxia may supervene when both vocal cords are fixed in the median position, while the voice is lost if the vocal cords are retained in the cadaveric position. Pain on swallowing may be considerable in acute cases, but is not usually a prominent symptom.

In luxation of the arytenoid cartilage the laryngoscopic image is peculiarly distorted. It is usually associated with tumefaction and ankylosis, but in a few instances simple luxation has been recorded.



FIG. 80.

Luxation of the left arytenoid cartilage following perichondritis.

Diagnosis.—When we consider the numerous causes of ankylosis, the various complications that may be present, and the variable symptoms that may result, it will obviously often be a very difficult matter to determine whether ankylosis be present in a case or not.

In the great majority of cases of true ankylosis there is tumefaction around the crico-arytenoid joint, and thus tumefaction, with fixation of vocal cord, favours the diagnosis of ankylosis.

But in other cases no tumefaction may be present, especially in old-standing cases of vocal cord paralysis which have become



FIG. 81.

Ankylosis of the left crico-arytenoid joint on deep inspiration.



FIG. 82.

The same during phonation.

ankylosed from prolonged immobility. Thus ankylosis may exactly imitate in appearance simple paralysis of a vocal cord.

Yet generally, in incomplete unilateral ankylosis of a vocal cord, there is some perceptible inward movement of the arytenoid cartilage on phonation, while in simple paralysis of one vocal cord, without ankylosis, the arytenoid cartilage in the paralysed side is displaced by the sound and over-adducted arytenoid during phonation. Attention to this point sometimes enables one to distinguish between a paralysed vocal cord and simple fixation of the cord from disease implicating the crico-arytenoid joint, for in the latter case the healthy arytenoid approaches, but *does not displace*, that on the affected side.

Nevertheless, it must be borne in mind that a paralysed vocal cord becomes more or less fixed in course of time from simple disuse: the diagnostic point has therefore greater positive than negative value.

Treatment.—The treatment of the inflammatory condition in acute cases is similar to that employed in acute laryngitis.

In the chronic cases very little can be done to remove the inflammatory thickening around the joint, and if the symptoms are not inconvenient or dangerous, it is better to leave well alone and avoid the risk of setting up fresh inflammation by local interference. But when dyspnœa is a prominent symptom, owing to laryngeal stenosis, it becomes necessary to resort to tracheotomy or to some method of overcoming the narrowing of the glottic chink, by means of intubation or Schrötter's bougies, continued over a long period. Dilatation affords some hope of relief in cases of spurious ankylosis. When true ankylosis is attended with marked dyspnœa, tracheotomy is the only remedy, and should be performed as early as possible lest the patient be carried off by a rapid increase in the respiratory difficulty.

The same remarks apply to luxation with ankylosis; but in simple luxation it may be possible to replace the arytenoid cartilage, provided the luxation is fairly recent. Cheval states that he was able to reduce a luxated arytenoid by means of a strong faradic current, a double electrode being applied to the posterior wall of the larynx so as to tetanise the inter-arytenoid and posterior crico-arytenoid muscles.

CHAPTER IX.

CHRONIC INFECTIVE DISEASES.

SYPHILIS—TUBERCULOSIS—LEPROSY—LARYNGEAL AND TRACHEAL
STENOSIS—INTUBATION.

SYPHILIS OF THE LARYNX.

Inherited Syphilis of the larynx occurs: (1,) within the first few months of life, when it usually takes the form of laryngeal catarrh, or the milder manifestations of secondary acquired syphilis; (2,) about puberty; in this later form, tertiary manifestations may be encountered.

Congenital syphilitic subglottic stenosis of the larynx may occur in young children. Sometimes syphilitic ulceration of the trachea just below the larynx arises in association with inflammatory syphilitic swelling, causing dyspnœa.

Acquired Syphilis of the larynx assumes the characters described as secondary and tertiary (one case of primary chancre is reported by Moure), but "secondary" manifestations may arise and keep on recurring many years after the primary sore; and on the other hand, "tertiary" forms may be met with rarely within a few months of the initial lesion. While this statement applies especially to laryngeal syphilis, it is likewise true of faucial and nasal syphilis.

Syphilitic manifestations in the larynx take the form of:—

- (1,) Simple catarrh (Lewin's erythema).
- (2,) Papules (condylomas; mucous patches).
- (3,) Diffuse infiltration and superficial ulceration.
- (4,) Gumma and deep ulceration.
- (5,) Epithelial outgrowths, neoplasms.
- (6,) Perichondritis.
- (7,) Cicatrices and fibroid contractions.
- (8,) Paralysis.

Of these the first three groups are more usually encountered in the earlier or so-called "secondary" stages, in which also superficial ulceration is common; the remainder belong to the later or, "tertiary" stages.

Syphilitic Catarrh scarcely differs from simple non-syphilitic catarrh, except in its persistency. It may occur as soon as six or eight weeks after the primary sore, and is often associated with general secondary manifestations. The history and the occurrence of syphilitic lesions in other parts generally enable one to make a correct diagnosis. An erythema with patches of red on the vocal cords is suggestive of syphilis. Unless the vocal cords are implicated, simple erythema of the larynx causes no symptoms.

Mucous Patches are not often seen. In fact, early syphilitic disease of the larynx gives rise to such slight symptoms that it is frequently unsuspected, and for this reason the tertiary form of the disease, with more pronounced symptoms, more often comes under the notice of the physician.

The circumscribed grey thickening of the infiltrated epithelium may occur on the epiglottis (especially on the lingual surface), the ary-epiglottic folds, ventricular bands, posterior commissure, or on the vocal cords. They are generally single, or if multiple are not symmetrical. Superficial erosions, yellow, oval, circumscribed, and surrounded by an areola, may follow denudation of the softened epithelium, especially in professional voice users. The symptoms are hoarseness, and slight expectoration (*Plate XVIII, Fig. 1.*)

Diffuse Infiltration leading to tumefaction of the epiglottis, inter-arytenoid fold, or vocal cords may cause peculiar hoarseness, the "raucous" voice, and sometimes dyspnoea. The infiltration may break down, forming chronic superficial ulcers or may undergo fibroid transformation.

Gummas are sometimes seen before breaking down as smooth, red or yellowish, defined swellings; generally single and occupying the epiglottis (its margin or the laryngeal surface), the ary-epiglottic folds, posterior wall of the larynx, or the ventricular



FIG. 83.

Bilaterally symmetrical superficial ulcers on the vocal processes in secondary syphilis.

bands: or they may be infraglottic, or tracheal. Very rarely they commence in the perichondrium. When about to break down the centre becomes yellow and ulcerates. The whole gumma then rapidly disintegrates from the centre towards the periphery, and a characteristic syphilitic ulcer results. (See p. 159.)

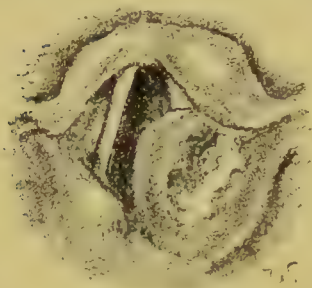


FIG. 84.

Breaking down gumma of the left ary-epiglottic fold.

Perichondritis generally occurs in association with gummas, either by deep extension of the infiltration or, more rarely, the infiltration may be seated primarily between the perichondrium and the cartilage. In the latter case especially, necrosis, and destruction or exfoliation of the cartilage is liable to follow.

In 1880 Semon drew attention to a sclerosing form of perichondritis in which a fibroid change occurred without any breaking down or caries of the cartilage. This form is chronic and persistent, and leads to marked stenosis and deformity.

Neoplasms or mammillated outgrowths are often found projecting from the posterior commissure. They resemble those found in tubercular laryngeal disease, but consist of proliferated epithelium. They are sometimes associated with ulcerations (see *Plate XVIII*,



FIG. 85.

Syphilitic out-growths in the inter-arytenoid space.

Fig. 3.)

Fibroid Contraction of the larynx follows diffuse infiltration, which undergoes the fibroid metamorphosis, and gives rise to marked stenosis. Cicatricial contraction is as characteristic of syphilitic ulceration of the larynx as of the fauces. A web may be formed between the cords, as in the case illustrated.

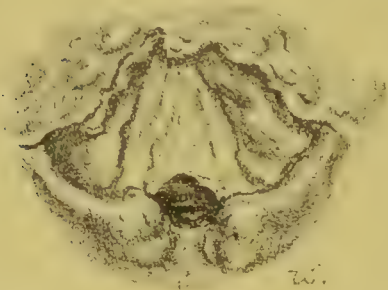


FIG. 86.

Extensive destruction of the epiglottis, and formation of cicatricial web between the vocal cords. The result of tertiary syphilis.

Paralysis. *Fixation of the Cords* from ankylosis of the arytenoid cartilages is a frequent consequence of fibroid thickening

in this region. *True paralysis* of the vocal cords from nuclear disease, syphilitic neuritis, or from pressure on the motor fibres of the nerves to the larynx in the brain, or in the nerves at the base of the brain, may occur, and is sometimes the earliest physical sign of a diffuse syphilitic lesion of the brain.

Symptoms.—It will be obvious that the symptoms of laryngeal syphilis must vary according to the nature and seat of the lesion. The most remarkable feature common to nearly all is their freedom from pain ; but while the absence of pain is remarkably characteristic of syphilitic lesions, here, as in the fauces, it is not safe to rely on this feature too absolutely. A gumma of the epiglottis may cause considerable dysphagia, or if on the posterior surface of the cricoid cartilage, much pain may be felt on deglutition. The voice is generally affected—to what extent depends, of course, on the extent and seat of the disease. In the earlier manifestations the chief symptom is hoarseness, and the peculiar raucous voice of advanced syphilitic laryngeal disease often enables one to make a diagnosis before examining the larynx. There is often great difficulty in distinguishing the cord at all in cases where the ventricular bands have been much altered and infiltrated, and with the vocal cords ulcerated and more or less completely gone, or cicatrised and adherent to the ventricular bands.

Treatment.—The general treatment of syphilitic lesions in the throat is practically the same as for syphilis occurring in other regions, and therefore need not be discussed at length. The method of mercurial inunction recommended by Von Zeissl is often more satisfactory than exhibition of mercury by the mouth. Twenty grains of mercurial ointment are rubbed in daily into various parts of the body for seven days, the series being preceded and followed by a warm bath. This process is repeated four or five times, according to the necessities of the case. Careful cleansing of the teeth, and the use of astringent and antiseptic mouth washes are indicated to avoid mercurial stomatitis. It is necessary, moreover, to emphasise the importance of not adhering rigidly to routine methods for the so-called secondary and tertiary forms of the disease, for we shall often find it desirable to administer iodide of potassium in the secondary form, and mercury in the later manifestations.

Mucous plaques, however, often give much trouble before they disappear. As a rule, the application of solid nitrate of silver,

or of the saturated solution, carefully limited to the affected areas, should be made every alternate day until the patches begin to resolve—combined, of course, with internal antisyphilitic remedies. Where there are cracks or erosions, or when pain is present, Browne has usefully substituted iodine and carbolic acid as a local pigment. In obstinate cases McBride advises painting with chromic acid grs. x ad ʒj).

The foul ulcers of tertiary syphilis may require a gargle of chlorate of potash, or some other mild antiseptic; or the appli-

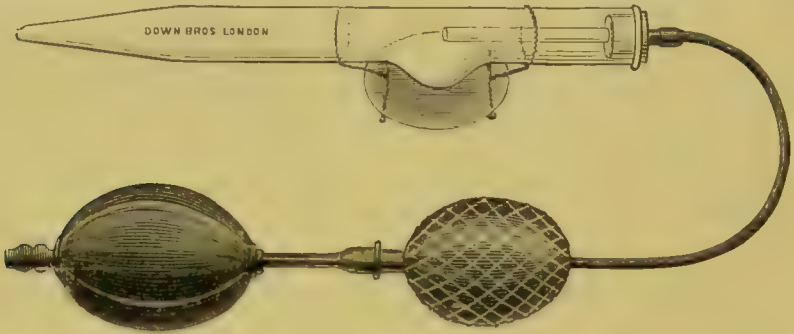


FIG. 87.

Calomel Fumigator for laryngeal and nasal syphilitic ulcers and for calomel fumigation in membranous croup.

cation of a solution of sulphate of copper or carbolic acid. Iodide of potassium should be given internally in daily doses of 60 grains from the commencement; in the later manifestations, gummatous infiltration and ulceration and perichondritis should

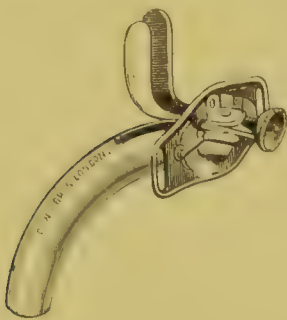


FIG. 88.

Störk's Tracheal Cannula and Dilator for chronic laryngeal stenosis.

be rapidly brought under control by free administration of the iodides, combined with mercury. If ulceration is extensive and progressive, the calomel fumigator may be used with advantage. Stenosis of the larynx, if chronic, may necessitate tracheotomy or intubation. As a rule tracheotomy is to be preferred, as syphilitic stenosis is so liable to recur after dilatation by Schrötter's bougies or intubation tubes. If the stenosis be due to fixation of the vocal cords, it may be possible to

mechanically dilate the glottic opening, and to keep them diverged by means of Störk's dilator till they have become fixed in the new position; or one cord may be excised.

O'Dwyer has urged the advantages of intubation, and several of his cases have yielded most brilliant results. Often only small tubes can be passed at first, but after leaving these in for twelve or twenty-four hours it is generally possible to introduce larger ones, and eventually obtain a permanent stretching of cicatricial tissue.

Cicatricial web formations should be divided by the cutting dilator, and intubation tubes worn till the edges have healed, so as to obviate re-union and re-formation of the web.

TUBERCULAR LARYNGITIS.

The records of Brompton Hospital show that tubercular disease of the larynx is found in at least 50 per cent. of all patients dying of chronic pulmonary tuberculosis; whilst about 20 per cent. of patients suffering from phthisis manifest signs of tubercular laryngeal disease (P. Kidd). Willigk, in 1,317 autopsies in cases dying of tuberculosis, found the larynx implicated in 237. Histological examination would probably reveal even a larger percentage of laryngeal involvement.

Etiology and Pathology.—Primary laryngeal tuberculosis may undoubtedly occur, but the affection is usually secondary to tuberculosis of the lung, although physical examination may not reveal the lung disease, for the physical signs, in any but advanced cases, tend to be obscured by the throat affection. It occurs more frequently in men than in women, in the proportion of about 2 to 1.

The tubercular affection probably originates by direct infection by the sputum passing over it. A long standing or frequently recurrent catarrhal laryngitis often precedes the true tubercular deposit, and possibly predisposes to infection by weakening the resisting power of the tissues; or from the invasion of the epithelium by micrococci small superficial erosions occur, and afford a means of entry for the bacilli as described by Lake.

The penetration of tubercle bacilli through the epithelial cells without previous erosion by cocci has been demonstrated in the larynx by Jon. Wright. Much light has been thrown on the pathogenesis of laryngeal tuberculosis by the researches of Jobson Horne on a large number of larynges which presented none of the usual naked-eye signs of the disease. He showed that the earliest changes were found in the lymphatics, consisting

(a,) in proliferation of the endothelial cells forming the wall of the lymph space ; (b,) the division and fusion of the adjacent and divided cells ; and (c,) the separation of this plasmodial mass as a giant cell. As in all stages tubercle bacilli were found in the earlier period of the process lying amongst the endothelial cells, he was of opinion that once having gained entry into the lymphatic ducts, they caused the subsequent cell proliferation by their irritation.

These observations account for the fact that the regions most rich in lymphatics are the common sites of tuberculous infiltration and ulceration, viz., the inter-arytenoid region, the posterior third of the cord, the ventricular band, and the epiglottis (especially the petiolus). Alongside with this cell proliferation, a proliferation of subepithelial blood-vessels takes place, and in the muscles changes take place in their fibres in an early stage, these changes being clinically brought into evidence by an early functional failure which is mainly myopathic.

We may distinguish three stages in the development of the tubercular lesions :—

(a,) The *first stage* is accompanied by pricking sensations in the throat, irritability of the fauces and larynx, and general languor. The mucous membrane of the larynx and pharynx is usually remarkably pale even at this stage before any local deposits of tubercle can be observed. But pallor is not always

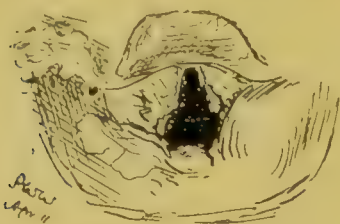


FIG. 89.

Tuberculosis of the larynx, with ulceration of the epiglottis and infiltration of the ary-epiglottic folds and ventricular bands.



FIG. 90.

Turban-shaped infiltrated epiglottis and pear-shaped arytenoids from tubercular infiltration.

present, and if present is often transient, while not rarely the larynx may be simply congested, or the affected area may even be brick-red.

During this early stage, the vocal cords often lose their pearl-grey sheen and become whiter and more opaque, and their movements in phonation and inspiration may be lagging and

Syphilis and Tuberculosis of the Larynx.



FIG. 1.

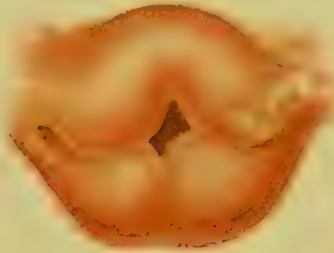


FIG. 2.



FIG. 3.



FIG. 4.

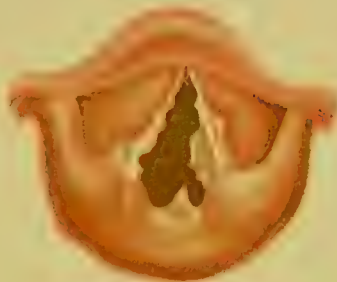


FIG. 5.



FIG. 6.



FIG. 7.

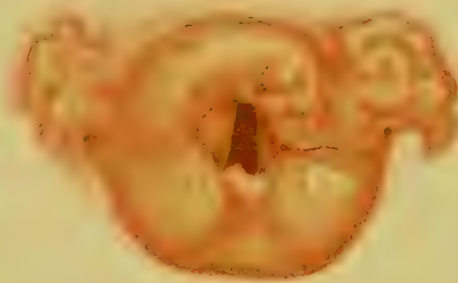


FIG. 8.



FIG. 9.

FIG. 1.—Secondary superficial syphilitic ulceration of the vocal cords.

FIG. 2.—Acute inflammatory swelling, with superficial syphilitic ulceration of the epiglottis.

FIG. 3.—Tertiary syphilis. The vocal cords have been the seat of former syphilitic ulcers, which have cicatrised with partial union of the cords. The posterior boundary of the glottis is occupied by syphilitic mammillated outgrowths, rather more succulent than usual.

FIG. 4.—Superficial tubercular ulceration of the ventricular bands and of the right vocal cord. The general pallor of the mucous membrane of the larynx is very noticeable.

FIG. 5.—The indurative type of laryngeal tuberculosis. The arytenoid regions are diffusely infiltrated. Outgrowths are seen projecting from the arytenoid space and under surface of the left vocal cord. Both cords are ulcerated; the ventricular bands also show irregular infiltration. The patient died within a few months, with both lungs breaking down.

FIG. 6.—Laryngeal tuberculosis, with secondary oedema of the arytenoid regions.

FIG. 7.—Laryngeal tuberculosis, with general infiltration.

FIG. 8.—The same, advanced. The epiglottis is breaking down, and extensive ulceration has occurred on the anterior surface along the lateral glosso-epiglottic folds. In a syphilitic patient.

FIG. 9.—Laryngeal tuberculosis. The whole of the larynx is anæmic and infiltrated with tubercular deposits, forming characteristic anæmic sausage-shaped swelling of the epiglottis, pear-shaped arytenoids, and swollen ventricular bands.

sluggish. A crenated appearance of the mucous membrane of the inter-arytenoid fold is commonly observed in tuberculous patients who subsequently develop laryngeal tuberculosis.

(b.) After a variable period, sometimes of long duration, the *second stage of deposition* manifests itself. Generally, pale smooth swellings of the inter-arytenoid fold, arytenoid regions, or epiglottis appear, at first unilaterally, soon becoming bilateral, till the turban-shaped pale greyish epiglottis and the pear-shaped swelling of the arytenoids present the characteristic aspect of tuberculosis of the larynx (see *Plate XVIII, Fig. 9*). In some cases miliary tubercles may be observed forming in the pink swollen mucous membrane, as greyish yellow points which are obviously lying beneath the translucent mucous membrane, and which rapidly increase in size and number till by coalescence they form the typical pale grey infiltrations more commonly seen. In other cases the disease remains more or less localised to limited areas of the larynx, and assumes an indolent, less characteristic, or even an indurative form resembling lupus, syphilis, or carcinomatous infiltration.

(c.) The *third stage of ulceration* rapidly succeeds the tubercular infiltration. Tubercular ulcers are superficial, often multiple, with irregular "mouse-nibbled" edges, difficult to define from the surrounding pale grey infiltration, and covered with pale greyish-white *débris*. They tend to spread slowly and superficially rather than deeply. There is no attempt at cicatrization in tubercular disease (see *Plate XVIII, Fig. 4*).

Again, indurative tubercular laryngitis, with slight tumefaction and little ulceration, may persist for many months, and somewhat suddenly take on the more typical and rapidly progressive characters of laryngeal tuberculosis. This indurative form often presents the mammillated proliferations already alluded to (see *Plate XVIII, Fig. 5*).

Paresis or paralysis of the vocal cords is not uncommonly due to the associated laryngitis inducing paresis of the thyro-aryte-

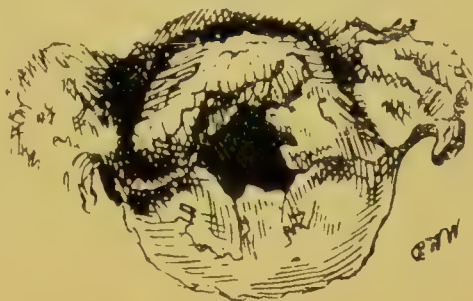


FIG. 91.

The same as FIG. 90, but two months later, showing more extensive infiltration and rapidly advancing ulceration of the tubercular deposit.

noidei muscles, or to waxy degeneration of the muscles. True paralysis of a vocal cord may be the result of pressure on the recurrent laryngeal nerves; thus the nerve may be implicated in pleural thickening at the apex of the lung, generally the right, or an enlarged gland may press on either nerve.

While the epiglottis and the arytenoid regions are the favourite seats of tubercular disease, the ventricular bands or inter-arytenoid folds may be the first part of the larynx to be affected, the other regions remaining healthy for a considerable time. Eventually the perichondrium of the cartilages may become involved, with resulting perichondritis and caries or necrosis.

The vocal cords may be attacked first, becoming red and swollen, the tumefaction breaking down and forming ulcers on the cords, without any other part of the larynx being affected. Ulcers on the vocal cords are common in advanced tubercular disease of the larynx.

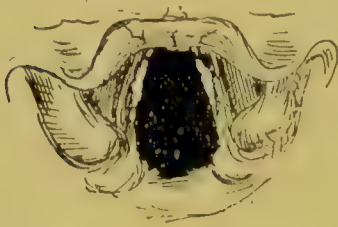


FIG. 92.

Tubercular ulceration of the vocal cords.

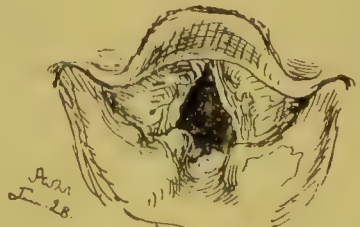


FIG. 93.

Mammillated tubercular outgrowths in the inter-arytenoid space, with infiltration and ulceration in other parts.

Tubercular neoplasms occur in the form of (1.) granular or mammillated hyperplasias; (2.) papillary excrescences; or (3.) smooth, defined neoplasms.

Fungous, villous, or mammillated outgrowths are sometimes observed, sprouting frequently from the inter-arytenoid fold, or from the vocal cord, or rarely in any other region; the outgrowths sometimes cover the whole arytenoid fold. It should be remembered that very similar outgrowths of a non-specific nature may occur in long-standing simple laryngitis, and in syphilis, though Störk and Mandl consider that their presence in the inter-arytenoid region is almost pathognomonic of tubercle.

Smooth, rounded tuberculous neoplasms are of rare occurrence

in the larynx. Payson Clark, who collected forty-two cases, states that these growths appear beneath an unbroken surface, and rarely ulcerate. They are usually sessile, and the colour of the overlying mucous membrane is not much changed. They resemble fibromas or papillomas, and may be strongly indicative of malignant disease in other parts of the body.

Cases of *prolapse of the ventricle* of Morgagni have been described as occurring in phthisical patients and others. The ventricle cannot prolapse, and the appearance is due to a tumefaction or neoplasm in the mucous membrane of the ventricle, extending beyond the laryngeal aperture of the sacculus. Doubtless it is sometimes a tuberculous process.

The Symptoms of laryngeal tuberculosis will obviously vary greatly according to the character and extent of the lesions. Only slight hoarseness may be noticed in early cases, but when more advanced the most prominent features are cough, pain on deglutition associated with rapid loss of flesh, a hectic temperature and great prostration.

The constant worrying cough is due to the extremely irritable condition of the larynx, the amount of expectoration depending mainly on the extent to which the lungs are involved.

Pain on swallowing is present when the epiglottis is infiltrated, and is often agonising when it has undergone ulceration. Perichondritis, of the cricoid especially, is attended with pain on swallowing. The swelling of the epiglottis or arytenoid regions may prevent closure of the glottis, so that fluids find their way into the larynx and bring on attacks of painful coughing. As a consequence of the intense pain and difficulty attending swallowing, patients will often refuse almost all food, while the saliva and secretions are allowed to accumulate and dribble from the mouth.

The degree of hoarseness depends on the implication of the vocal cords, while inter-arytenoid swelling prevents their approximation, and results in aphonia; or the arytenoid cartilages may be fixed by the tumefaction, with consequent hoarseness or aphonia.

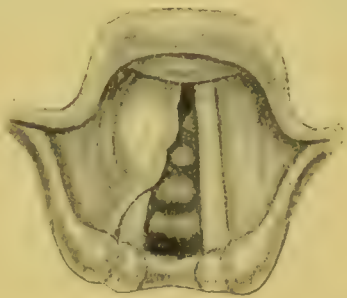


FIG. 94.
Tubercular tumour of the ventricular band. AVELLIS.

Diagnosis.—The diagnosis of laryngeal tuberculosis is not usually difficult, the pallor of the mucous membrane, the characteristic tumefaction of the epiglottis and arytenoid regions, the superficial, mouse-nibbled aspect of the ulcerations, and the associated pulmonary and general indications of tubercular disease, and especially the presence of tubercle bacilli in the sputum, leaving little room for doubts. But in other cases the diseased areas are restricted, and may resemble simple catarrhal laryngitis, and there is a curious tendency for the physical signs of early pulmonary tuberculosis to be masked or obscured when the larynx is involved. A unilateral congestion of one vocal cord or arytenoid cartilage, or persistent localised areas of cedematous tumefaction, or even a frequently recurrent laryngitis without adequate exciting cause, would suggest some serious affection.

The diseases most liable to confusion with laryngeal tuberculosis are lupus, syphilis, and malignant disease. For their differential diagnosis reference may be made to the table on page 159. But it should always be remembered that tuberculous disease may co-exist with either syphilis or malignant affections of the larynx.

Prognosis.—The acuteness of onset and development varies greatly in different cases, and at different periods in the same case, but the affection of the larynx is always an extremely grave complication of tuberculosis. The onset in some cases is very insidious, and the infiltration and ulceration may occur without any symptom but hoarseness being complained of; but when we remember that 50 per cent. of patients dying from phthisis show laryngeal complications, the importance of attending to any laryngeal symptoms in pulmonary consumption is obvious. When the disease is localised, and is not associated with advanced pulmonary tuberculosis, the results of local treatment are hopeful. The worst and most rapidly fatal cases are those in which diffuse miliary tubercles appear rapidly in different parts of the larynx..

Treatment.—The general treatment of laryngeal tuberculosis of course includes the general treatment of pulmonary tuberculosis, but one or two points require special notice. Firstly, the patient should be strictly enjoined to use the voice as little as possible; he may speak in a low whisper instead of phonating.

All irritants, such as tobacco, strong alcoholic drinks, highly seasoned food, dusty occupations, etc., should be avoided; food should be cool, bland and soft, and alcoholic stimulants well diluted. The internal administration of codeine or morphine, or both combined, in the form of a linctus or by sucking pastils, has an excellent effect in relieving pain and irritation in the larynx, and thus largely aids in securing the all-essential rest, and enabling the patient to take food and to sleep. Of course these sedatives must not be pushed so far as to prevent expectoration altogether, but sufficiently to allay the useless and constant irritating cough which is nearly always a distressing symptom.

As regards the question of residence, the general consensus of opinion is opposed to sending patients with laryngeal affections to high altitudes. There may be exceptional cases, of course, but we have the high authority of Theodore Williams for regarding laryngeal tuberculosis as a contra-indication for the rarefied dry air of the higher Alps. In fact, patients who are too far advanced for the active treatment of the laryngeal complication are, as a rule, better kept at home; or, if they are able to settle abroad, the northern African sea-board, or the Riviera in Europe, will afford the most suitable climatic conditions. On the other hand, if active therapeutical measures are to be carried out, it is better to postpone the question of residence abroad till the laryngeal disease has been arrested. Experience has shown that very beneficial results may generally be expected, in all but advanced cases of pulmonary tuberculosis from the open-air, hygienic, medicinal, and dietetic treatment under medical supervision in sanatoria; and laryngeal implication is no contra-indication for such rational methods which, *combined with local treatment of the larynx*, have yielded most favourable and encouraging success. There is some need, however, for a warning against relying too completely on the effect of sanatorium treatment apart from local measures.

In discussing *local treatment*, we may divide cases into three classes :—

(a,) Those in which no definite tubercular deposit is observed, but only a catarrhal laryngitis, localised or general. For such, local measures consist in subduing the pain and cough, if present,

by menthol or cocaine, either applied locally or by a spray or pastil. Thus :—

℞	Menthol -	-	-	-	gr. xij
	Eucalyptol	-	-	-	℥ xij
	Terebene	-	-	-	℥ xij
	Cocainæ -	-	-	-	gr. vj
	Ol. vaselini	-	-	-	ʒj

To be used in the oil atomiser before meals when the cough is troublesome.

Or,

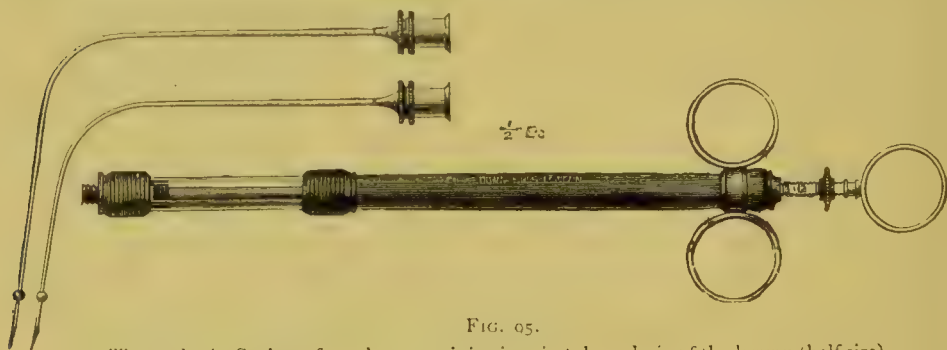
℞	Menthol -	-	-	-	gr. $\frac{1}{4}$
	Codeinæ	-	-	-	gr. $\frac{1}{4}$ vel
	Morphinæ	-	-	-	gr. $\frac{1}{2}$
	Acid. citric	-	-	-	gr. $\frac{1}{2}$
	Saccharin elix.	-	-	-	q. s.
	Fiat pastil				

To be dissolved slowly in the mouth ; three or four may be used daily if required.

A pastil, or the use of the spray at night, will often ensure a good night to the patient.

(b.) Those in which a definite tubercular deposit has occurred in the larynx.

If only a local tumefaction without ulceration is present, I have obtained excellent results from the injection into the affected mucous membrane of a few minims of guaiacol ; or a



The author's Syringe for submucous injections in tuberculosis of the larynx (half size).

solution of perchloride of mercury in water and glycerin, 1 in 1000 ; or aristol, 2 per cent, in almond oil. A similar plan of treatment has been employed by Krause, Chappell, and Donelari.

As the result of these injections, the pain is almost always diminished in the course of about twenty-four hours, and in

several cases the mischief was arrested after repeated injections. Krause removes such tumefactions with his double curettes, and applies lactic acid; Gougenheim employs a very similar instrument, especially for arytenoidectomy, in cases attended with dysphagia. If the epiglottis alone is affected, it may be possible to extirpate the implicated area by the snare or by cutting forceps, and thus arrest the disease.



FIG. 96.

Case of tuberculous laryngitis treated by submucous injections and curettement.



FIG. 97.

The same larynx four months later.

If ulceration has occurred, lactic acid should be well rubbed into the ulcerated surface. If these milder measures, combined with vocal rest and sedatives internally, do not prove sufficient, I generally follow Heryng's practice of first curetting the tubercular necrotic tissue before rubbing in the lactic acid. Krause removes the tubercular deposit by means of his double sharp

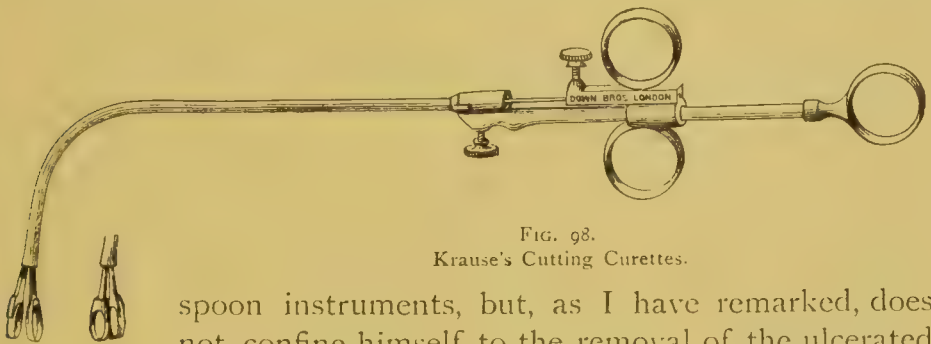


FIG. 98.

Krause's Cutting Curettes.

spoon instruments, but, as I have remarked, does not confine himself to the removal of the ulcerated deposits.

The lactic acid solutions should be strong (50 to 80 per cent.), and must be applied by practised hands only to the affected parts of the larynx. In some cases such strong solutions of this powerful irritant are not well borne; the strength should then be reduced to 15 or 20 per cent.

Of course, all these procedures require a previous application of a 10 or 20 per cent. solution of cocaine, till the larynx is completely anæsthetised.

Ulcers on the vocal cords should be simply rubbed with the lactic acid, which is applied by means of a small pledget of cotton wool firmly wound on a laryngeal probe. The patient should be confined to a warm room, and be directed to suck ice after the operations, and only to take cold and perfectly bland liquid food. Pain should be relieved by morphine, either hypodermically or combined with cocaine hydrochlorate in minute doses in the form of a pastil.

If any active inflammation or perichondritis is present, *curettement* and the application of lactic acid are generally contra-indicated, for the disease must be quiescent before such radical procedures can be undertaken with safety or any prospect of success. The ulcerated surface may be cleansed with a spray of perchloride of hydrogen and morphine, and a small quantity of powdered orthoform mixed with gum acacia insufflated. The patient thereby obtains relief from pain for some hours, and is enabled to swallow soft food without discomfort.

In favourable cases the tubercular ulcers may be made to cicatrise by the methods just described, and cases are recorded, and have occurred in my own practice, in which the larynx has remained free from tubercular disease for years.

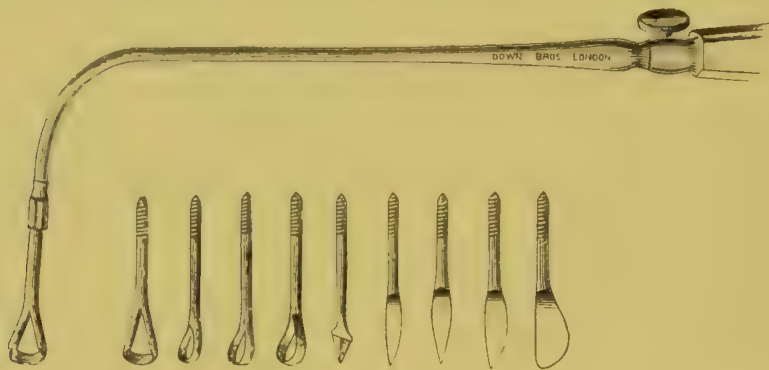


FIG. 99.

Heryng's Laryngeal Curettes.

My own experience agrees with Heryng's views, *vis.*, that *curettement* is especially indicated: (1.) In cases of circumscribed, slowly developing tubercular infiltrations, even though they may show no tendency to break down. The object of the operation

here is to prevent the further destruction of a vital organ by destroying the centre of infection. Even somewhat advanced lung disease and a certain degree of fever, so long as it is not of a hectic character, cannot in all cases be regarded as contra-indications. If the tubercular infiltration is confined to the posterior wall of the larynx, as is most frequently the case, then an early and as radical as possible removal of this can bring the process to a standstill for months and years, and restore the functions of the larynx : (2,) In many diffuse infiltrations which run their course with special violence, and even when the general condition is relatively unfavourable—*e.g.*, excessive dysphagia due to inflammatory swelling and ulceration of the epiglottis or posterior wall of the larynx—for the pain can be alleviated in the quickest manner possible for a considerable time, though, of course, healing cannot take place.

The chief contra-indications Heryng considers to be (a,) Advanced phthisis of the lungs with hectic and wasting ; (b,) Diffuse miliary tubercle of the larynx, or rather of the larynx and pharynx ; (c,) All cachectic conditions ; (d,) Severe stenosis of the larynx caused by inflammatory swelling of the affected parts. In these cases tracheotomy must be performed as soon as possible ; (e,) Patients exhibiting fear and nervous excitability, mistrust of a physician, and especially in those whose condition promises little hope of recovery. [*Journ. of Laryng.*, Aug., Sept., 1893, Aug., 1894.]

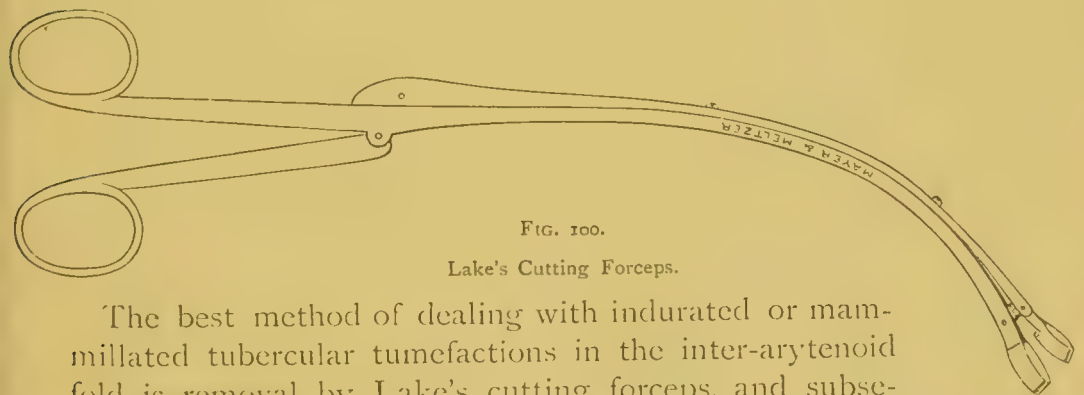


FIG. 100.

Lake's Cutting Forceps.

The best method of dealing with indurated or mammillated tubercular tumefactions in the inter-arytenoid fold is removal by Lake's cutting forceps, and subsequent application of lactic acid. Heryng has only seen severe hæmorrhage twice, in each case after removing a tumour-like hard false cord. He therefore considers it advisable to destroy hard tumour-like tubercular infiltrations of the false cords by

means of electrolysis or the galvano-cautery. The subsequent treatment of the wounded surface with lactic acid after *curette-ment* depends on whether we have succeeded in removing all the diseased parts.

The other question demanding consideration is the possibility of an outbreak of general tuberculosis being hastened or excited by a local operation in the larynx. Hitherto I have not witnessed such an event, though Lermoyez and Sokolowski each report an instance, a connection rather difficult to prove.

Scheppegrell has recorded favourable results from cupric interstitial cataphoresis. He uses spherical electrodes of chemically *pure* copper, one-eighth to one-fourth of an inch in diameter, applied to the diseased areas by means of an insulated handle. The copper electrode, connected with the positive pole, is applied to the affected areas in the larynx, the negative pole being connected with a large dispersing electrode to the nape of the neck. The larynx is previously sprayed with cocaine or eucaine in solution, and a galvanic current of not more than 4 milliampères should be thrown in and reduced by using a rheostat. The method is stated to be applicable to all cases of laryngeal tuberculosis, and, when properly applied, gives rise to no pain, irritation, or subsequent reaction. It caused granulations and infiltration to diminish, and ulcerations to heal, and apparent cure in one of the recorded cases.

Other methods often afford relief, *e.g.*, a solution of 12 or 15 per cent. of menthol with 2 to 4 per cent. of guaiacol dissolved in olive oil and injected through the larynx into the trachea by means of a special syringe, as advocated by Downie.

(3.) Those cases which are associated with acute inflammation or perichondritis, or in which the rapidly advancing lung affection has greatly weakened the patient, can only be treated by palliative remedies such as local applications of morphine, cocaine, or menthol, or the internal administration of morphine or codeine, etc. The relief afforded by such means should not be forgotten, and considerable benefit to the patient often results from the possibility of swallowing without pain, and the increased amount of sleep that anodyne sedatives induce.

Tracheotomy is rarely called for, and should only be performed when dyspnoea is urgent. It is generally to be preferred to intubation, since it is only resorted to in cases where an intubation tube would not be well tolerated.

LEPROSY OF THE THROAT AND NOSE.

The throat and nose are frequently affected in patients suffering from the cutaneous form of this disease, but it has never been known to occur primarily in the respiratory tract.

It is unnecessary to enter into the vexed question of the etiology of this affection beyond remarking that the lesions are due to the *lepra bacillus* so closely resembling the bacillus of tubercle, that at one time it was even regarded as a modified form of tuberculosis.

Unna's view, that the leprosy bacillus inhabits the lymphatics and not the cells of the tissues, has been corroborated by Bergengrün, whose sections show that the so-called globi and lepra cells are simply cross-sections of bacillary thrombi in the lymphatics, and that the giant-cells are due to endothelial proliferation around the bacillary thrombi. The primary lymphatic infection and the formation of giant cells by proliferation of the lymphatic endothelium, both in leprosy and in tuberculosis of the larynx is a point of great pathological interest, and explains the important part played by the lymphatics in the further dissemination of these infective diseases in the body.

Leprosy of the larynx may assume the tuberculated or nodular form, or, very rarely, the anæsthetic variety. In either case the onset is extremely insidious, owing to the painless nature of the affection, and patients will sometimes declare that they have nothing the matter with the throat, when examination reveals that it must have been developing for a considerable period.

Tuberculated Leprosy of mucous membrane passes through three stages. In the first stage the uvula and soft palate, in which small nodules extending backwards in the middle line are



FIG. 101.

Leprosy of the Larynx (WAGNIER).

usually first observed, become red and velvety in appearance, the neighbouring tissues becoming affected by continuity or by separate foci of disease, so that the nasal mucosa, the epiglottis and aryteno-epiglottidean folds become red, velvety, thickened, and hard, like the soft palate and uvula, and appear as though coated with varnish. The mucosa of the arytenoids often becomes thickened in the shape of thick globular masses. Bergengrün states that the epiglottis, especially the petiolus and the regions above and below the anterior commissure, are favourite seats of infection. The epiglottis is often curved upon itself, and may be so thickened that the interior of the larynx cannot be seen. At this stage epistaxis frequently occurs, and the patient may complain of shortness of breath, a sense of tickling and dryness in the pharynx, larynx and nose.

In course of time, the red, hard infiltration becomes soft, and the tissues somewhat œdematous, the redness soon giving place to pallor till the affected regions are uniformly pale, resembling the anæmia of tubercular deposit: and when the infiltration and cellular elements become absorbed, the tissues appear, as Mackenzie puts it, as though they were infiltrated with tallow. The hard palate is often involved and nodular.

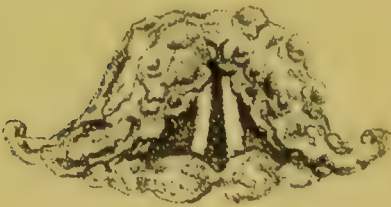


FIG. 102.

Leprosy of the Larynx (G. MACKERN).

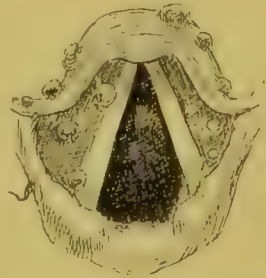


FIG. 103.

In the second stage, the characteristic tubercles appear, first as small, whitish-yellow, elevated nodules, varying in size from a pin's head to a split pea, isolated, or in chains or groups. They may remain stationary for years. Ramon de la Sota y Lastra, whose valuable article in Burnett's system is based on a very extensive experience, states that on the appearance of the nodules the uniformly swollen condition of the membrane disappears.

The third stage of ulceration may be reached, but the patient often succumbs to the general affection ere the nodules break down. Ulceration along or below the vocal cords, or in the ventricular bands, is common, and may be extensive. The epiglottis may be completely destroyed. The ulcers are rounded, elevated above the surrounding mucous membrane, and are compared by De la Sota to syphilitic mucous patches. The cartilages of the larynx are sooner or later involved and become necrosed, and may be exfoliated.

In leprosy of the nose there is a curious trilobed external appearance, and when the mucous membrane is involved by extension from the *alæ nasi*, the chief symptom is nasal obstruction. But when the nodules ulcerate, the stench of the sanious watery discharge is often intolerable. The cartilaginous septum becomes perforated, and, with the *alæ nasi*, may be destroyed by extensive ulceration. Cervical lymphatic enlargement is usual, but it is indolent and never suppurates.

The earliest indication of the extension of leprosy to the upper respiratory passages is alteration in the voice, which at first nasal in quality, soon becomes shrill. Later in the disease, hoarseness or aphonia results from the implication of the vocal cords. Dyspnoea is rarely very marked, though in a few cases tracheotomy is required for the stenosis of the larynx resulting from œdema or from the nodular infiltration.

The sense of smell and taste is usually blunted or lost early, and the loss of sensation is peculiar and complete. De la Sota has found that there is often complete anæsthesia without analgesia.

The *anæsthetic form* of leprosy is stated by Hillis, of Demerara, never to occur until the cutaneous disease has existed for at least five years. The mucous membrane is pale, the velum palati paralytic, and the affected regions are anæsthetic.

Diagnosis.—Leprosy of the throat and nose requires to be differentiated from syphilis, tuberculosis, lupus, and cancer. There is rarely any difficulty in making the diagnosis, in that leprosy is never a primary affection of the upper air passages, but always secondary to, or at least concurrent with, cutaneous leprosy, though a leprous patient may be affected with cancer, lupus, or syphilis of these regions.

Treatment. — Mackern has successfully treated laryngeal leprosy in the early stages by galvano-cauterisation of each nodule in succession, but in most cases coming under observation treatment can only be palliative, and is especially called for in the ulcerative stage when alkaline and antiseptic solutions may be useful, or for the relief of dyspnœa by tracheotomy. De la Sota has noticed improvement under the application of a 1 per cent. solution of resorcin, and of iodoform dissolved in ether, and from touching the diseased areas with a 10 per cent. solution of chloride of zinc.

STENOSIS OF THE LARYNX AND TRACHEA.

Etiology. — The various conditions liable to result in LARYNGEAL STENOSIS have been discussed already in connection with the descriptions of laryngeal diseases, but it will be useful to summarise and group these conditions and to contrast the symptoms with those due to tracheal stenosis. Stenosis of the larynx may be due to:—

(1.) Infiltration of the tissues: (*a*.) in the course of acute inflammatory affections, or from injuries of the larynx; (*b*.) cedematous exudation in renal disease, from the internal administration of iodide of potassium, etc.; (*c*.) by tuberculous, lupous, leprosy, or gummatous deposit.

(2.) False membrane in croup, diphtheria, scalds, etc.

(3.) Neoplasms, benign or malignant.

(4.) Bilateral abductor paralysis of the vocal cords from whatever cause arising.

(5.) Cicatricial contraction following syphilis, lupus, enteric fever, perichondritis, wounds, etc., or congenital web formations.

(6.) Foreign bodies.

TRACHEAL STENOSIS. — Various pathological conditions may result in stenosis of the trachea, which, if considerable, may cause symptoms resembling laryngeal stenosis, or when too inconsiderable to cause pronounced dyspnœa, may be confused with morbid conditions of the larynx or lungs.

The causes of tracheal stenosis may be either (1.) extra-tracheal, or (2.) intra-tracheal.

External causes act by compressing and flattening the trachea, the most common being enlargement or growths of the thyroid or thymus glands, aneurysms, mediastinal tumours; and, more

rarely, pericardial effusion, dilatation of the left auricle, large tuberculous glands, and large pleural effusion in children.

Internal causes act by swelling or infiltration of the mucous membrane, by the encroachment of new growths, or by constriction of the tracheal wall, and include foreign bodies, œdema (inflammatory or urticarial), pemphigus, membranous tracheitis, tuberculosis, lupus, leprosy, rhino-scleroma, neoplasms, syphilitic gumma, syphilitic cicatricial contraction, and stricture following tracheotomy; of these, the last four alone are at all common.



FIG. 104.



FIG. 105.

Laryngoscopic appearances in cases of tracheal stenosis due to compression by enlarged thyroid gland. *Fig. 104* shows the usual scabbard stenosis, and *Fig. 105* the oblique stenosis due to compression by one lobe only of the thyroid gland.

The symptoms of tracheal stenosis vary according to the nature of the cause and the degree of respiratory obstruction. Dyspnœa is usually the predominating symptom, and the patient's attitude is often characteristic, the head being thrown forwards and the chin depressed so as to relax the trachea. The voice is clear but weak from diminished force of expiration from the unfilled chest. With marked stenosis the peculiar characteristic "mewing" inspiratory sound is developed, especially on exertion; and the usual harsh bronchial inspiratory and expiratory sounds may be replaced by soft low breath sounds, though more usually by a well-marked stridor, both inspiratory and expiratory, audible over the trachea and upper part of the sternum, or they may disappear entirely.

When the stenosis is due to inflammatory exudations, neoplasms, or ulceration in the trachea, hæmoptysis (spurious) is commonly associated with cough and expectoration of mucus. External compression is more likely to be associated with involvement of one or both pneumogastric or recurrent laryngeal nerves, with resulting laryngeal and bronchial spasm and brassy cough, or laryngeal paralysis.

In the earlier stages of intra-tracheal conditions causing stenosis, the symptoms often resemble bronchitis, asthma, or stridulous laryngitis.

Diagnosis.—Wherever possible investigation of the cause of dyspnoea must comprise a careful laryngoscopic examination, and thus by excluding all intra-laryngeal causes, a strong presumption of tracheal or main bronchial stenosis may be established. But it must be borne in mind that a unilateral or even bilateral paralysis of the larynx may be the result of compression of the recurrent laryngeal or main vagus trunks by morbid conditions which concurrently cause tracheal compression and stenosis. Unilateral laryngeal paralysis in an otherwise healthy larynx, never causes dyspnoea during rest in the adult, in the child it may do so; but bilateral abductor paralysis often causes attacks of urgent or even fatal dyspnoea. But the recognition of co-existing tracheal stenosis is important because it would be useless to perform a tracheotomy or laryngotomy when the essential cause of dyspnoea is further down. In laryngeal stenosis the head is retracted, whereas in tracheal stenosis it is usually thrown forwards; in laryngeal stenosis, from any cause except paralysis, the voice is hoarse or aphonic, whereas in tracheal stenosis the voice is clear though weakened; in laryngeal stenosis, stridor is usually pronounced during inspiration only, whereas in tracheal stenosis it is both expiratory and inspiratory, though the latter is most marked.

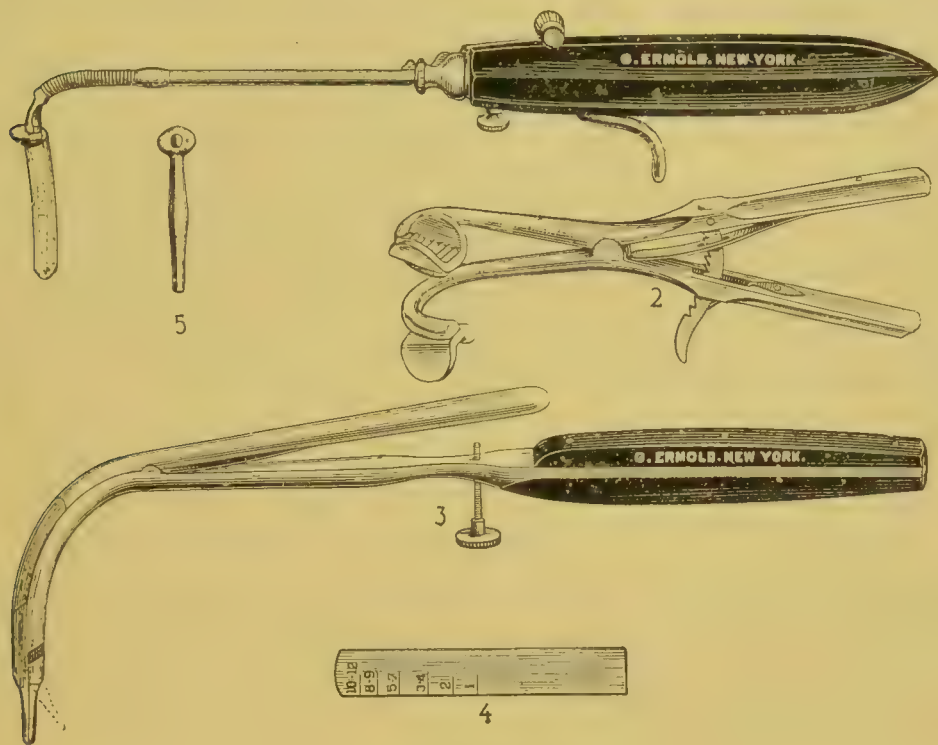
Treatment.—As a rule very little can be done to relieve tracheal stenosis, owing to the cause being nearly always within the chest. In a few cases, however, the compression of the trachea is accessible to surgical relief, either by a low tracheotomy or by the removal of a compressing growth. Complete relief has followed rapidly after removal of the isthmus of the thyroid gland or of the lateral lobes causing tracheal stenosis. But compression by an enlarged thyroid gland may be amenable to medical treatment alone, and, as an instance in point, I may add that the patient from which Fig. 104 was taken was completely relieved by the administration of thyroglandin.

When the stenosis is due to aneurysm, the only hope of temporary relief is absolute rest in the position most comfortable to the patient (usually sitting up in bed with the head and shoulders thrown forwards). The possibility of the stenosis being due to

syphilitic gumma should never be forgotten, for in these cases relief may be afforded by the prompt and free administration of appropriate antisyphilitic remedies.

INTUBATION OF THE LARYNX.

O'Dwyer's operation is in most cases the best method for the relief of laryngeal stenosis. His tubes are made of gilt metal, and vary in length from one and a half to two and a half inches



FIGS. 106-110.

O'Dwyer's instruments for intubation.

for children of different ages. A flange at the upper extremity of the tube rests on the glottic margin with the tube *in situ*, the rest of the tube being below the vocal cords.

For intubation, the little patient having been firmly wrapped in a light blanket, the arms being included, is usually held sitting upright on the nurse's lap, and the mouth held open with the gag by an assistant. But whenever the child is very weak or the heart's action feeble, it is advisable to intubate with the patient lying flat on the back, a method which is scarcely more difficult than with the patient sitting up.

The operator, facing the patient straight, hooks forward the epiglottis with the *left* forefinger. The selected tube is then quickly introduced on the obturator held in the *right* hand, and a sliding rod on the obturator handle then disengages the tube, which should be kept *in situ* while the obturator is withdrawn.



FIG. 111.

To show the method of intubating the larynx.

The tube is now in position, but with a silk loop-thread attached to the flange (previous to introducing), by which it may be withdrawn for re-introduction, should it have been inadvertently passed into the œsophagus. When the tube has been successfully inserted, the patient coughs violently a few times. The loop need not be cut and withdrawn, and at any rate should be left "until it is evident that all obstruction to the breathing has been overcome and

no partially detached false membrane is in the trachea below the tube. The thread at first acts as an inciter to cough, which is desired; ordinarily ten minutes is sufficient" (Northrup). The larynx and trachea soon tolerate the foreign body, the urgent dyspnoea is at once relieved, the patient generally dropping off into a calm sleep.

Extraction of the tube is more difficult. For this, specially constructed forceps with closed blades are guided by the *left* forefinger tip into the upper orifice of the tube, opened so as to hold the tube firmly, and then withdrawn. Neither introduction nor extraction of the tube should occupy more than fifteen seconds, respiration being of necessity temporarily suspended during their performance. The position of the child is the same as for introduction.

The advantages of the operation over tracheotomy are :—

(a.) Its simplicity and painless nature, well illustrated by the fact that a child, seven years old, who had on former occasions experienced intubation and extraction at my hands, sat up and permitted me to extract the tube without being held, or in any way restrained. On this account we can resort to intubation much earlier than the more formidable operation of trache-

otomy ; and avoid all "cutting," to which the parents sometimes will not consent.

(*b*.) In children under five years of age, the percentage of recoveries in published cases is considerably higher than after tracheotomy, while above this age the percentages of recovery are about the same.

(*c*.) The intubation tube is worn much more comfortably than a tracheotomy tube ; in fact, when *in situ* it can seldom be felt by the patient at all.

(*d*.) Coughing is more effectual ; expectoration, therefore, more perfectly performed.

(*e*.) The respired air passes through the natural passages.

(*f*.) No anæsthetic is required. Cocaine is generally very useful.

Accidents and difficulties that may occur with intubation are:—

(1.) Pushing of false membrane into the trachea on introducing the tube. This occurred only three times in O'Dwyer's first two hundred and nine cases. As the thread is left attached till this danger is passed, it is a simple matter to remove the tube and allow the loosened membrane to be expectorated.

(2.) Coughing out the tube, with immediate return of dyspnoea—a rare occurrence.

(3.) Asphyxia, from blocking of the tube by false membrane. This can only occur in an extremely feeble patient, as the tube is always expelled by a vigorous cough when, as occasionally happens, it gets blocked up. I have known the tube coughed up and swallowed in two cases without ill effects.

Northrup states that the symptoms of loose membrane are : (*a*.) croupy character of cough, the tube being in ; (*b*.) flapping sound ; (*c*.) sudden obstruction to out-going air, especially during coughing. When this condition is suspected, the thread should not be cut, but looped over the ear, protecting it along the cheek with adhesive plaster. Short tubes (for loose membrane) have been devised for these cases. They are short hollow cylinders of large calibre, and being short will not push down the tracheal membrane. As they have no retention swell, it is necessary to use the largest possible size.

(4.) Asphyxia from œdema above the tube and closing the orifice is a possible, but improbable, occurrence, inasmuch as the flange rests on the ary-epiglottic folds.

(5.) Ulceration of the larynx just above the ventricular bands, or at the cricoid ring, from an ill-fitting tube. When the mucous membrane lining the cricoid cartilage becomes infiltrated and cedematous, it swells towards the centre, and dyspnoea results. As O'Dwyer pointed out, if a tube is forced into the constriction, ulceration and sloughing of the tissues are almost certain to occur, and in some instances necrosis of the cricoid cartilages has occurred. When difficulty arises from sub-glottic stenosis at the cricoid ring, the narrowest part of the respiratory passages, the tube meets with obstruction here, and may even creep back, "like an oiled cork in a bottle." The only safe course then is to use a tube of small calibre.

(6.) The inspiration of particles of food and consequent pneumonia. This is the only untoward accident that I have experienced in many cases, and it can be obviated by using nutrient enemata while the tube is in.

(7.) Careless and forcible introduction may produce a false passage.

(8.) The special danger attending extubation is the tearing of tissues from opening the forceps widely at the side of, instead of within, the tube.

(9.) Asphyxia from prolonged unskilled attempts at introduction or extraction is avoided by making several attempts, if necessary, rather than one prolonged one.

(10.) Another danger in extubation arises in some cases from the deposit of calcareous matter which takes place on metal tubes, but not on rubber tubes, when they have been left many days *in situ*. The concretions may cause laceration, and subsequent ulceration, of the mucous membrane during removal of the tube.

Intubation should be performed early so as to prevent engorgement of the lungs, and their partial collapse consequent on prolonged dyspnoea. Children who can understand should have nourishment in the form of thickened fluids, which they should "gulp" slowly. Casselberry's method of making the child swallow with the head low down and on its face has the effect of making the child gulp in this way. For those who cannot do this, nutrient enemata alone are preferable. The tube should not be left in for more than five days; speaking generally after three days it should be removed and replaced as often as neces-

sary. I have generally found it desirable to remove and clean the tube at least once every other day, if this can be accomplished without disturbing the patient to any great extent. When a tube has to be removed and replaced several times it is well to follow O'Dwyer's plan of taking a rubber tube and immersing it in a solution of hot gelatin containing 5 to 10 per cent. of powdered alum, and then introducing the tube with a covering of alum gelatin. Fischer, while recommending this plan, has also found good results following a similar use of a 10 per cent. solution of ichthyol in gelatin. Temporary aphonia sometimes persists for a few weeks after the removal of the tube.

As regards recent statistics of intubation and tracheotomy, it will be enough to refer to the statistics given on page 137. With these may be compared the results of the collective investigation of the Deutsche Gesellschaft für Kinderheilkunde, of cases treated in pre-antitoxin years.

In four years 1,324 cases of primary diphtheria with laryngeal stenosis were treated by intubation, with 516 cures (39 per cent.). The method was only applied in cases of severe stenosis. It is applied to all degrees of age, and in the youngest children. Of secondary diphtheria, following measles and scarlet fever, pneumonia, etc., 121 cases were treated, with 27 cures (22 per cent.). In the first year of life 93 cases were treated, with 13 cures (14 per cent.); in the second year of life 295 cases, with 92 cures (32 per cent.). Of 1,324 cases of intubation, in 242 cases it was necessary to perform secondary tracheotomy, with 20 cures (7 per cent.); 58 cases with secondary diphtheria and secondary tracheotomy all died.

Of 1,118 cases treated by tracheotomy, 435 were cured (39 per cent.). Of 42 cases of secondary diphtheria, 11 were cured (26 per cent.). In primary diphtheria both methods gave exactly the same results, *i.e.*, 39 per cent. cures. In the first two years of life the results of intubation are better than those of tracheotomy.

I would add that it is impossible to draw comparisons between the relative advantages of intubation and tracheotomy from a simple comparison of results as exemplified by statistics, for whereas tracheotomy is seldom resorted to except in extreme cases, intubation is, or ought to be, undertaken as soon as

laryngeal obstruction has manifested itself. It is also impossible to conceive that the large array of cases tabulated by several observers in any way represents the class of case that would have been deemed suitable for tracheotomy. We must rely rather on the general impression, as to the relative merit of these alternative procedures in those who have had wide experience in both of them. But I may urge the advantages of intubation as affording a ready and effective means of tiding the patient over the period of dyspnœa that is usually relatively short when antitoxin has been given early. Intubation has certain disadvantages, as well as many advantages over tracheotomy, but one of the greatest disadvantages in tracheotomy is the difficulty that is so often encountered in removing the tube, owing to the presence of synechiæ or granulations, after the patient has recovered.

CHAPTER X. *NEOPLASMS OF THE LARYNX.*

BENIGN NEOPLASMS--MALIGNANT NEOPLASMS.

UNDER neoplasms in the larynx are included the benign and malignant new growths, but not the vegetative forms of laryngeal syphilis and tuberculosis, nor the epithelial formations termed pachydermia laryngis, nor of course, inflammatory deposits, although clinically they constitute laryngeal tumours.

BENIGN NEOPLASMS.

Etiology. -Benign growths of the larynx are fairly common ; they are most frequently met with between the ages of twenty and forty, but are nearly as often encountered in young children, and may occur at any period of life, sometimes arising in old age. They are somewhat more frequent in the male than in the female sex, but the difference between the sexes in this respect is by no means so marked as in the occurrence of malignant disease.

The actual causes of these neoplasms are most indefinite, though it is usual to regard chronic laryngeal catarrh and over-use of the voice as important factors. But the very frequent occurrence of benign laryngeal growths in young children, and their occasional existence in the newly-born and in deaf-mutes, argue strongly against these current views. Moreover, though chronic laryngitis is a common affection, no one has observed new growths actually arising during its course, if we except the little inflammatory nodules on the cords called "singers' nodules" ; nor is chronic laryngitis a pronounced feature in the majority of the cases of new growths.

The various forms of benign growths occurring in the larynx, in order of frequency are as follows : papilloma, fibroma, cystoma, myxoma, adenoma, lymphoma, lipoma, angioma, ecchon-droma, and those composed of thyroid gland tissue. Of these,

only the first three forms are at all common, all the others being exceedingly rare.

Papilloma.—This, the commonest variety of laryngeal new growths, is met with at all ages, but especially in young adults.



FIG. 112.

Multiple papillomata of the larynx.

It is almost the only form seen in young children. Papillomata may be single, but are generally multiple, varying in size from a millet seed to a walnut, of a delicate pink granular surface, with an almost characteristic cauliflower-like uneven surface. They are usually pedunculated, less often sessile, firm, elastic, and do not readily bleed (see *Plate XIX, Figs. 4 to 7*). In histological structure, according to Virchow, all the

papillomata are warts, consisting of epithelial cells, with a small formation of connective tissue.

The favourite site is the free border or upper surface of the middle or anterior half of the vocal cords (especially the right cord), or the anterior commissure. Unless multiple, they are less frequently seen on the ventricular bands, ary-epiglottic folds, or epiglottis. Sometimes they project from the ventricle of Morgagni or the inter-arytenoid fold, and in this position they might be mistaken for syphilitic or tubercular granulomas, or even for a "pachydermia verrucosa" by the inexperienced. Benign papillomas are rare in the inter-arytenoid fold, while this is a favourite seat of epithelioma, a fact that should be remembered, especially in patients past middle life. Unlike epitheliomata, their area is distinctly limited, they do not infiltrate the surrounding tissue, and, moreover, are practically never seen in the posterior third of the vocal cord. Yet early epithelioma of the larynx may very closely resemble a benign papilloma (see *Plate XX, Fig. 4*).

Fibrous Polypus (the so-called fibroma) is a fairly common variety of tumour almost confined to adult life. Fibromata are generally single, sessile, with light pink or cherry-red, smooth surface, and are mostly found occupying the upper surface of the middle or anterior half of a vocal cord, and vary in size



FIG. 113.

Fibroma of the vocal cord.

from a millet seed to a walnut. They may be pedunculated, and if large may present an indented uneven surface resembling a papilloma. O. Chiari has shown that the growths consist of the same tissue as the vocal cords, and originate in inflammatory thickening of the vocal cord from congestion; hence they are vascular and contain cavernous blood spaces. Serous infiltrations and hæmorrhages are common, especially in the softer growths. Small œdematous fibromas may very closely resemble myxoma both in laryngoscopic appearance and in microscopic examination.

Cystoma may result from obstruction in the duct of a muciparous gland, or distension of a lymphatic vessel, and consequently generally occurs where these are plentiful, especially the epiglottis. Louys states that they may also owe their origin to the thyroglossal duct or to the pyramidal lobe of the thyroid gland, and Chiari has observed cysts developing in the interior of laryngeal polypi. They are rarely found also on the ventricular bands, or growing from the ventricle, the arytenoid region, or from the vocal cords (see *Plate XIX*, *Fig. 1*).

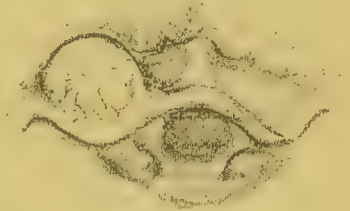


FIG. 114.
Cyst on the lingual surface of the epiglottis.

They are smooth, tense, globular, semi-translucent, and covered with light red or grayish-pink mucous membrane, and if considerable in size, blood vessels are seen coursing over the surface. They may be simple or multilocular.

Angioma is a rare form of tumour, of characteristic aspect, generally unilateral and single, occurring in the sinus pyriformis, or on the ventricular bands, vocal cords or epiglottis, and rarely exceeding a filbert nut in size (see *Plate XIX*, *Fig. 8*. The colour of the growth should have been a deeper purple).

Myxoma usually occurs on the vocal cords as a small, smooth, pink or grayish-white, sessile, translucent tumour; unilateral and distinctly localised. When pedunculated it is rather a fibromyxoma in structure, and then may present a mammillated surface and resemble a papilloma in appearance.

Echondroma is a very rare form of growth, arising generally from the cricoid cartilage. Echondromata have been found in connection with the epiglottis, thyroid and arytenoid cartilages.

They are firmly attached, hard sessile growths, presenting a smooth surface of irregular outline and covered with healthy mucous membrane.

Lipoma and adenoma are exceedingly rare forms of laryngeal neoplasm. According to Gerhardt only ten cases of lipoma had been reported up to 1896. Two years later Hinkel recorded a case of fibro-lipoma which recurred several times after removal.

The so-called "*Prolapse of the Ventricle of Morgagni*" so closely resembles a neoplasm of the larynx in its clinical aspects that it is convenient to allude to it here. A smooth, pink, lobulated, supra-glottic mass, either unilateral or bilateral, is seen resting on the vocal cords and corresponding to the opening of the sacculus. The sacculus cannot really prolapse, and the appearance is due to a tumefaction or deposit in the lining mucous membrane. It is most frequently associated with phthisis pulmonalis.

Symptoms.—Pain is hardly ever experienced, the symptoms being almost confined to impairment of voice, and a greater or less degree of obstructive dyspnoea, varying, of course, according to the situation and size of the growths.

In a certain number of cases no symptoms whatever occur, or they are so slight as to cause no discomfort to the patient. But even a small growth occupying the free border of a vocal cord, or the anterior commissure, may very greatly impair the voice or produce complete aphonia.

Cough is sometimes a marked symptom, particularly in young children with papillomata, in whom it may be croupy in character, especially as in them the growth is liable to excite some laryngitis and glottic spasm.

Treatment.—It is usually better not to operate on benign papillomas in children unless they cause respiratory obstruction, for: *firstly*, they are very liable to recur again and again after removal (this tendency to recurrence disappears after about two years); *secondly*, they sometimes disappear spontaneously. If the voice is much impaired and the growth is fairly accessible, it may be well to remove it by intra-laryngeal operation, but only after trying the local application of salicylic acid dissolved in absolute alcohol (2 to 5 per cent. as recommended by Dundas Grant), for a considerable period, a method that has proved very fairly successful. Bryson Delavan has recorded a cure from the

Benign Growths in the Larynx.



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

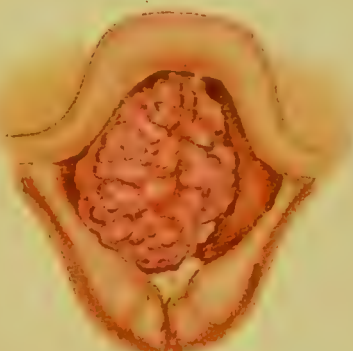


FIG. 5.



FIG. 6.



FIG. 7.



FIG. 8.

FIG. 1.—Cystoma of the epiglottis. Symptoms practically nil.

FIG. 2.—Fibroma, cured by removal through the mouth.

FIG. 3.—Fibroma of the vocal cord, subglottic.

FIG. 4.—Papilloma laryngis during inspiration, the growth hanging down between the cords.

FIG. 5.—The same, during phonation, when the growth was extruded above the glottis.

FIG. 6.—Papilloma laryngis:

FIG. 7.—Benign growth attached to the under surface of the left vocal cord of an adult male. On attempted vocalisation the growth projected upwards, preventing closure of the vocal cords. Removal completely restored the voice.

FIG. 8.—Angioma of the larynx.

*P. Wilson Williams
ad naturam del.*

Scott & Fensholt, Edin^r

local application of absolute alcohol simply, in the form of a spray six times daily. There were large papillomatous masses on the vocal cord, and they steadily improved till in six months they had disappeared. He has also noted several instances of marked improvement by this method of treatment. Others have obtained improvement from a course of full doses of arsenic. When these measures fail or if dyspnoea arises, removal by intra-laryngeal operation should always first be attempted by an expert laryngologist. This failing, if the dyspnoea calls for relief, tracheotomy should be performed. In a considerable number of cases the growths have decreased and eventually disappeared from this operation alone. If after a prolonged interval of not less than twelve months, the growths persist and intra-laryngeal extirpation has been tried and has failed, then thyrotomy may be performed and the growth carefully removed. The great objection to removal by splitting the thyroid cartilage is that even if skilfully and successfully performed, there is a considerable risk of permanent impairment of the voice remaining. This may sometimes be due to the difficulty in exactly hitting the middle line in splitting the thyroid cartilage, but in other cases it is due to the formation of granulations or of a cicatrix in the anterior commissure during the process of healing, and may be quite unavoidable. At the same time we must remember that papillomata in children may rather rapidly increase in size and number, and an operation for its relief may become necessary.

After removal of a papilloma, it is well to paint the seat of the growth with salicylic acid in alcoholic solution every few days, and to administer small doses of the proto-iodide or bin-iodide of mercury for some weeks. The excellent results obtained with *Thuja occidentalis* in the treatment of warts by Kaposi and others suggest the trial in cases of laryngeal papilloma unsuitable for operation, as well as with a view to prevent recurrence after removal.

In adults there is less liability to recurrence, and if the voice is impaired, or other symptoms are present, we should not hesitate to remove a papilloma. It has been maintained by some laryngologists that operative interference increases the liability to the occurrence of malignancy in benign growths, but any doubts on this point have been set aside by Semon's elabo-

rate investigation, which proved that the liability of a benign growth to become malignant, while always excessively remote, is diminished rather than increased by skilful removal.

Thus, of 10,747 cases of innocent laryngeal growths observed by 107 laryngologists, 8216 had been operated on intra-laryngeally; of these, in 33 cases malignant degeneration was reported, *i.e.*, 1 in 249 cases. (But critical examination of the individual cases renders it probable that only 1 in 685 cases exhibited malignant degeneration.) On the other hand a positively larger number of spontaneous degenerations in non-operated cases were reported, amounting to 1 in 211.

Fibromas and other benign growths should generally be removed, especially if that can be effected by intra-laryngeal methods, but it is sometimes better to leave them alone if there are no symptoms causing inconvenience. Fibromas and angiomas bleed freely, and if at all considerable in size it is safer to perform tracheotomy first, or to remove them by splitting the thyroid cartilage.



FIG. 115.

Mackenzie's Cog-wheel Exciser.

As regards the choice of operation, the decision must depend on the nature and situation of the growth as to whether we use the snare, *écraseur*, curette, forceps, the cautery or knife, or resort to an external operation. Of course all intra-laryngeal operations require that the larynx and fauces should be well cocaineised.

Very small pedunculated growths on the free edge of the vocal cords may occasionally be removed by Heryng's curettes, as advocated by Massei, while Dundas Grant's guarded forceps are both safe and suitable for growths on the free edge of the cords about the middle third, or on the glottic commissures. For all moderate sized growths I prefer my own combination forceps, which also appear to me to offer special facilities for the removal of minute growths, inasmuch

as one blade is stationary and may be held in contact with the growth while the other movable blade is made to close upon it. For the larger growths snaring, when feasible, is preferable to picking away with forceps.

The galvano-cautery must be used with the greatest caution and skill, and is rarely called for.

Of external methods, there are three alternatives: (a,) thyrotomy;* (b,) subhyoid pharyngotomy; (c,) laryngotomy.

It is claimed, by some operators, that the more complete removal of papillomas by the external operation lessens the liability to recurrence. Even if this be true—and it is at best only a question of degree, for recurrence does take place even after the most thorough removal by thyrotomy—there are the ever present risks of permanent vocal impairment and of some danger to life from the operation itself, and therefore should only be resorted to under the conditions



FIG. 116.

Dundas Grant's Safety Laryngeal Forceps, with antero-posterior, and lateral movement.

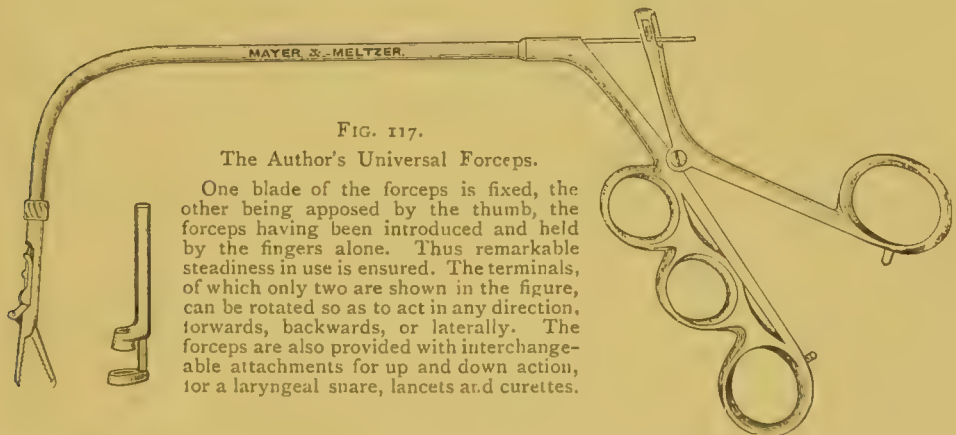


FIG. 117.

The Author's Universal Forceps.

One blade of the forceps is fixed, the other being apposed by the thumb, the forceps having been introduced and held by the fingers alone. Thus remarkable steadiness in use is ensured. The terminals, of which only two are shown in the figure, can be rotated so as to act in any direction, forwards, backwards, or laterally. The forceps are also provided with interchangeable attachments for up and down action, for a laryngeal snare, lancets and curettes.

already detailed. For subglottic growths involving respiratory embarrassment, thyrotomy or laryngotomy is sometimes unavoidable, but there are very few benign laryngeal growths

* *Syn.* Thyro-chondrotomy.

requiring removal which cannot be extirpated *per vias naturales*.

Cystomata should be seized with forceps. They always collapse, but the cyst wall being partly removed, they do not recur. *Angiomata* may be cauterised with a galvano-cautery point at a dull red heat.

MALIGNANT NEOPLASMS.

Malignant disease of the larynx, while by no means rare from the age of forty upwards, is less frequent than cancer in other regions or than benign growths of the larynx.

Etiology.—The predisposing and exciting causes of laryngeal cancer are as obscure as of cancer in other regions. Various predisposing causes, such as long-continued irritation, excessive use of the voice, have been regarded as causes, but with small justification. Apparently heredity has some influence, and men are more liable to the disease than women. The influence of age is seen in the rarity of laryngeal cancer below the age of forty, and the large proportion occurring between the ages of fifty and sixty, many arising between sixty and seventy, and a considerable number between forty and fifty, a period during which benign growths also are not uncommon.

Pathology.—Epithelioma is encountered with greater frequency than any other variety of malignant disease. Very rarely does the growth take the form of medullary cancer or scirrhus. Sarcoma of the larynx is very rare, but both round and spindle-celled forms occur as lympho-sarcoma, fibro-sarcoma and myxo-sarcoma.

Malignant disease of the larynx is almost invariably either primary or due to direct extension by continuity from neighbouring structures, scarcely ever does it arise by metastasis or secondary infection. This is due to the peculiar arrangement of the lymphatics of the larynx (see p. 16), which, though abundant, form a network of their own within the larynx proper without anastomosing with the lymphatics of the neighbouring tissues, anatomical conditions which obviously explain why malignant disease occurring in the interior of the larynx tends to remain localised for a long time without secondary glandular infection, and also why the larynx is so rarely the seat of secondary growths. On these facts rests the clinical importance of Kris-

haber's classification of laryngeal cancers into: (a,) *Extrinsic*, affecting the epiglottis, arytenoids, ary-epiglottic folds and pyriform sinuses; (b,) *Intrinsic*, comprising those arising on the vocal cords, ventricular bands, in the ventricles, and below the vocal cords within the borders of the larynx; (c,) *Mixed*, i.e., a combination of extrinsic and intrinsic, forming a large number.

Situation of growth.—Laryngeal malignant disease is more often primarily intrinsic than extrinsic. It usually commences on one of the true vocal cords, or in its immediate neighbourhood. Of the extrinsic cases, the posterior surface of the cricoid cartilage appears to be the most often implicated. But the malignant new growth may arise in any portion of the larynx.

Symptoms.—There is no single symptom which is characteristic of malignant disease of the larynx. Hoarseness or aphonia, spontaneous pain or odynphagia, dyspnœa, dysphagia, salivation, hæmorrhage, fœtor, glandular infiltration or cachexia, are the chief symptoms, but any one or all of these symptoms may be absent in the earlier stages, and there is no single symptom which may not be absent from first to last.

The symptoms vary of course with the size and seat of the deposit, and although there are certain features which, in most cases, suggest malignancy, it is often extremely difficult to form a definite diagnosis in the earliest stages, when it is a matter of very great importance for successful treatment.

Hoarseness.—The voice is almost always affected quite early in the disease, either from infiltration of the cord, or of the tissues around the arytenoid cartilages, or from implication of the recurrent nerves by enlarged lymphatic glands. A small tumefaction or a growth on the cord will cause a degree of hoarseness altogether disproportionate to its dimensions, partly owing to the resulting deformity of the vibrating margin, but especially on account of the impaired mobility of the cord which is almost characteristic of malignant disease.

Pain is not usually a prominent symptom till the later stages, and then, especially in extrinsic deposits, is often severe, darting up to the ear, and increased by swallowing,

Cough is not as a rule a marked symptom. Loss of appetite and a general cancerous cachexia often supervene early. Respiratory obstruction depends on the size and situation of the growth. In the intrinsic cases dyspnœa from obstruction often

becomes a prominent symptom. In the extrinsic the large size of the growth may cause glottic obstruction or, from implication of the recurrent laryngeal nerves or infiltration of the postici muscles, abductor paralysis may ensue and account for the dyspnœa.

Increased salivation (from reflex irritation) is generally present, and in cases with odynphagia the saliva collects and is allowed to dribble out of the mouth. When the growth ulcerates foul *débris* collects and imparts a sickly fœtor to the breath. Severe hæmorrhages are then liable to occur, slighter hæmorrhages are more usual in the earlier stages. Pain and dysphagia are rarely absent when ulceration has taken place, and are especially pronounced when the perichondrium becomes affected; abscess and necrosis of the cartilages may supervene, increasing the pain and dysphagia, and causing rapid loss of strength.

PHYSICAL SIGNS.—On the vocal cords, in its earliest stages, epithelioma appears either (1,) as a white or reddish-grey warty growth, generally single, and bearing a strong resemblance to a benign papilloma or fibroma; or (2,) as a diffuse unilateral infiltrating growth, with a red uneven surface; (3,) giving an uneven fringe-like margin to the affected vocal cord; or (4,) as a unilateral congestion. On the ventricular bands, in the ventricle, or on the ary-epiglottic folds it appears as a deep pink infiltration or in the form of a definite growth, with a coarsely mammillated or uneven surface. An epiglottic growth is often greyish or whitish-pink, and looks almost fibrous in texture, but with uneven surface; it may also take the form of a deep red, or dirty white, infiltrating growth.

The growth generally increases in size very slowly at first, but it soon extends rapidly and tends to ulcerate fairly early, or at least to become excoriated on the surface, and then readily bleeds. Growths originating on the cords or ventricular bands tend to spread backwards towards the interarytenoid fold and thence to the opposite side. Extrinsic growths extend more rapidly towards the pharyngeal walls, and in these deep ulceration is rarely long delayed. The floor of the ulcer is then covered with foul greyish muco-pus and *débris*, tinged with blood, giving a characteristic musty odour to the breath.

As the growth and ulceration extend, a secondary perichondritis often complicates the disease and renders the laryngoscopic appearance less characteristic.

From the arrangement of the lymphatics, extrinsic growths tend to earlier secondary infiltration of the lymphatic glands of the neck than is the case with intrinsic growths. In supraglottic deposits the glands between the larynx and carotids lying on the jugular vein at the level of the upper border of the thyroid cartilage, or beneath the greater cornu of the hyoid are often affected. In infraglottic cancer, while the glands of the neck escape, the lateral tracheal glands are often enlarged.

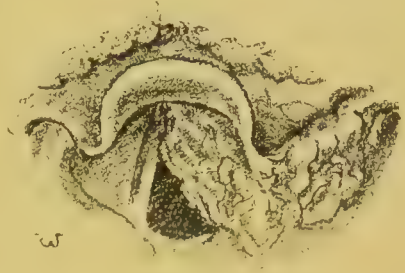


FIG. 118.

Epithelioma of the epiglottis. Laryngeal symptoms had been noticed for twelve months.

One of the earliest cases diagnosed and operated on has been recorded by Middlemass Hunt (see *Plate XX, Fig. 1*), and it admirably illustrates the value of some of the symptoms and signs which have just been described. The patient, a male, aged fifty-six, had been slightly hoarse for about a year. Laryngoscopic examination showed marked congestion of the left vocal cord, and on its edge, in the anterior third, was a small white, flat papilloma, which seemed to invade rather than to grow from the surface of the cord. There was perfect movement of both cords in phonation and inspiration. The growth was diagnosed as malignant from (1,) its white colour; (2,) its invasion of the substance of the cord; (3,) the soft, ulcerated appearance of its surface; (4,) the congestion of the cord on which it was situated; (5,) the age of the patient. The left cord was excised after thyrotomy, and the growth, which was no larger than a millet seed, presented the histological characters of an early epithelioma. The patient recovered with a good voice, and had remained free from recurrence some years later.

Medullary Cancer is difficult to distinguish from epithelioma of the larynx, except by microscopic examination. It is sometimes more vascular in appearance, it tends to involve the neighbouring lymphatic glands earlier, and to ulcerate more

rapidly and more deeply than epithelioma, and occurs mostly in the epiglottis.

Sarcoma may commence in the epiglottis or ventricular bands, or as an undefined, diffuse, infiltrating growth without definite location of origin. The tumour is generally single, and, if defined, is smooth, globular and semi-translucent, but it frequently takes the form of a greyish-pink, infiltrating growth, with smooth but uneven surface.

The rapidity with which it extends varies greatly in different cases, but it tends to remain unilateral, and in many instances the vocal cords escape, or are only implicated later in the disease.

The only case I have met with was a round-celled sarcoma, spreading from the thyroid gland to the larynx. It was observed as a subglottic infiltrating growth for three months before the voice was affected, and even then the tumour seemed for some time to be retarded in extension upwards by the corresponding vocal cord.

Diagnosis.—The main points to observe in the earlier stages of the disease are : (1.) The age of the patient ; (2.) The symptoms and laryngoscopic appearances ; (3.) The histological character of fragments of the growth ; (4.) The absence of symptoms pointing to other diseases liable to simulate laryngeal cancer, viz., syphilis, tuberculosis, gout, benign growths, pachydermia, lupus, laryngitis, perichondritis, and laryngeal paralysis or paresis.

Thus hoarseness coming on without some obvious cause, associated with congestion or tumefaction of *one* vocal cord, is strongly suspicious of syphilis, tuberculosis or gout, or if the patient be over forty, malignant disease. Catarrhal laryngitis is distinguished by being almost always bilateral, and further inquiry into the history and condition of the patient may suffice to differentiate the other affections.

In the early stages a warty growth on the vocal cords or ventricular bands resembles a benign papilloma, but appearing late in life a unilateral growth would excite suspicion, especially if firmly fixed and infiltrating the subjacent structures, with marked impairment of vocal cord movement, of a white or greyish-white colour, and attended with pain. Its occurrence on the posterior third of the vocal cords, or on the posterior commissure, would suggest malignancy.

Malignant Laryngeal Neoplasms.

FIG. 1.

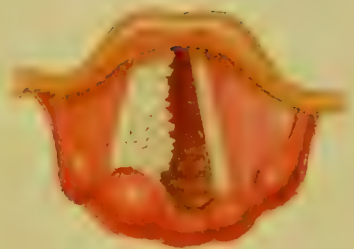


FIG. 2.



FIG. 4.

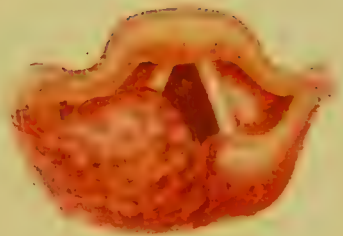


FIG. 3.

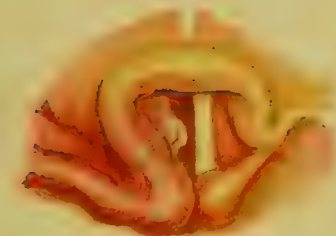


FIG. 5.

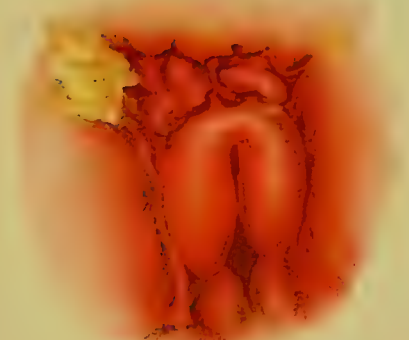


FIG. 6.

- FIG. 1.—Epithelioma of the left vocal cord. Case of Middlemass Hunt's successfully removed without recurrence.
- FIG. 2.—Cancer of the right vocal cord, showing the white mown-grass aspect so suspicious of malignancy in laryngeal growths. (For this drawing the author is indebted to Dr. J. W. Gleitsman, of New York.)
- FIG. 3.—Cancer of the larynx. An epithelioma is seen occupying the ary-epiglottic fold and extending to the arytenoid region and involving the ventricular band. It is bright pink and lobulated, but no portion had ulcerated when this drawing was made.
- FIG. 4.—Epithelioma of the larynx, showing two separate warty growths. (For this drawing the author is indebted to Dr. P. M'Bride.)
- FIG. 5.—Epithelioma of the epiglottis on the right side. Death seven months later.
- FIG. 6.—Epithelioma of the epiglottis, with resulting inflammatory infiltration of the epiglottis, simulating simple acute epiglottiditis. Death nineteen months later from the effects of the secondary deposits

Semon has emphasised the importance of a pronounced and increasing heaviness in the movements of the vocal cord, due to the infiltrating character of malignant growths, as a distinguishing feature, and this taken in conjunction with the accompanying circumstances, the age of the patient, abnormal sensations of pain, tenderness to pressure on the implicated side of the larynx, is very strongly suspicious of malignancy. If the growth originated in the ventricle and simply overlaps the cord, the vocal cord movements would probably not be impaired. The appearance of a fringy papillomatous margin along the greater part of the length of a vocal cord, or the imbedding of the cord in a dirty white mass, occurring in a patient past middle life is certainly to be regarded with grave suspicion.

A growth on the epiglottis or ventricular bands might be mistaken for a gumma. A gumma is very rapid in appearing, ulcerates very soon, is rarely painful, and generally yields to iodide of potassium. When, together with perichondritis, ulceration has occurred, it is most difficult to decide from the laryngoscopic appearance between a tertiary syphilitic lesion and a breaking down malignant growth. The symptoms and physical signs of tubercular disease and lupus are usually sufficiently definite to prevent their confusion with malignant disease in the larynx.

In doubtful cases a piece of the growth ought to be removed and submitted to the microscope. This should not be a very minute piece, as the evidence afforded may be equivocal; and the value of negative evidence must not be unduly trusted in coming to a decision as to the nature of the growth. Of course if the removed fragment yields definite and unmistakable evidence of malignancy, there is no room for doubt as to the diagnosis: but the negative evidence of a first microscopical examination cannot be held to exclude malignancy where the clinical symptoms and signs are distinctly suspicious. In the first place we must remember the possibility of the laryngeal growth being of a mixed character, or it may have a malignant base with a simple papillomatous or other non-characteristic surface. For these reasons it will often be desirable to remove a larger and deeper portion of the mass, and carefully and thoroughly to examine every portion. In other cases there may be no definite growth but merely a diffuse infiltration which will not afford a satisfactory fragment for histological diagnosis.

The difficulties in arriving at a definite conclusion as to the true nature of a tumour, even after a microscopical examination, is well exemplified by a case reported by Sokolowski. He removed a polyp from the left vocal cord, believing it was benign. Four years later the growth having recurred, it was considered malignant and cauterised. Again, eight months afterwards, another laryngologist operated on it, and from the microscopical examination and the clinical history concluded it was a benign adenoma. But Sokolowski then examined the growth he had removed five years before, and concluded that it was an adeno-carcinoma, and this subsequently proved to be correct, as the growth recurred in a manner which left no doubt as to its malignant character. Many similar experiences have occurred to various other laryngologists.

Unless the diagnosis is unequivocal, the patient should always be put through an anti-syphilitic course, but it must be remembered that malignant growths are often rendered less painful for a time by iodide of potassium. Moreover, it must be borne in mind that syphilis, tuberculosis, and various forms of benign growths may co-exist with malignant disease.

It is necessary, too, to remember that a gouty deposit in the larynx may resemble cancer in appearance and in its painful symptoms, and has been mistaken in more than one instance for *epithelioma laryngis*.

Prognosis.—The ultimate ending of untreated malignant disease of the larynx, of course, differs in no respect from the usual event of cancerous diseases, except that the risk of fatal asphyxia or persistent dysphagia may arise; in the one case from extension of intrinsic growth, in the other from involvement of the pharynx. But the rate of growth and rapidity of extension and glandular involvement, as we have seen, varies greatly according to the seat of the tumour, as well as with its pathological structure; growths confined to the vocal cords and ventricular bands being usually slow in development as compared with the extrinsic cases.

But the main question is whether the malignant disease can or cannot be successfully extirpated, and it is necessary to come to a decision with as little delay as possible, lest a case favourable for operative treatment be permitted to pass into the non-operable class.

The chief points for consideration are :—

(1,) Is the case a suitable one for operation? This depends on the seat and extent of the growth, the amount of glandular involvement, and the age and general condition of the patient.

(2,) What operation would be necessary, and what its risks, the chances of non-recurrence, and the functional result that it offers if successful?

In cases where the disease is limited to one vocal cord or its vicinity, the growth may be removed by splitting the thyroid. The results are very good in most cases, the mortality is small, and in nearly all cases a good voice remains. When the disease is mainly unilateral but has involved the opposite side, and yet remains intrinsic, the results of the more extensive removal required are still very good. Semon has obtained more than 82 per cent. of complete success in the foregoing classes of cases. But when the disease is extrinsic and involves the posterior surface of the cricoid, or the ary-epiglottic folds, the lymphatic glands are generally implicated, and in addition to extirpation of half the larynx usually necessary, which adds greatly to the risk of operation, the liability of recurrence is enhanced, while the voice is either lost or very imperfect. Nevertheless, in cases not too far advanced, operation in such cases may be justifiable, provided the patient be not too old, and feeble or broken in health.

When, however, both sides of the larynx are considerably involved, or when the disease has extended well over the posterior surface, or has involved the pharynx, complete laryngectomy and extensive removal of glands would alone offer any reasonable chance of non-recurrence. The operation by the Solis Cohen method may be accepted by the patient, but it should be explained to him that such an operation usually involves complete loss of voice and much permanent discomfort.

Finally, the general condition and age of the patient must always be taken into consideration. Those who are feeble and broken in health, or are the subject of pronounced lung disease, such as chronic bronchitis, emphysema, etc., or of chronic Bright's disease, and so forth, would obviously be unsuitable for operations which might be undertaken in those whose general health was fairly good.

Statistical results of the several operative procedures available have not been cited, for no tables of figures can in any sense indicate our present position: indeed, Bryson Delavan very correctly states that "there are no reliable statistics of operations for the surgical relief of malignant disease of the larynx." Success or failure depends so very largely on the individual skill of the operator, and there are not at present a sufficient number of operators of recognised position who have published exact details of all the cases they have operated on.

Treatment.—It is impossible as yet to lay down definite rules for the treatment of these cases, opinions being so divided as to the relative advantages of various operative procedures that have recently been introduced, in some cases with most brilliant results.

The choice lies between palliative treatment and radical operation. In the latter case we have five alternatives: (1.) extirpation through the natural passages; 2.) thyrotomy and excision; 3.) partial laryngectomy; 4.) sub-hyoid pharyngotomy; and (5.) total laryngectomy.

(1.) *Radical Operations.*—A few cases of successful extirpation through the mouth are recorded. It is available when the growth is pedunculated, circumscribed, and limited to the vocal cord; but only exceptionally, in early cases of intrinsic cancer, could this be attempted with any prospect of success. But when such immense issues are at stake, intralaryngeal operative treatment should never be recommended except in such cases where, owing to the general health or the refusal of the more certain prospect of cure by the patient, the external method is impracticable.

Sendziak's statistics of thirty-two cases of endo-laryngeal operation for laryngeal cancer gives 25 per cent. of favourable result (no recurrence for one year), and 40·7 per cent. of relapses, but statistics are not only worthless but misleading unless each case is considered on its own merits.

(2.) *Thyrotomy and Excision.*—Tracheotomy having been performed, a Hahn's or Trendelenburg tube is inserted in the trachea, time being given for the expansion of the sponge with the former tube. The thyroid cartilage is then laid bare by a median incision, and divided in the middle line, the two sides held apart. The extent of the growth is carefully noted, and, as pointed out by Semon, it will often be found to be much more extensive than the laryngoscopic appearance indicated. Billroth and Semon recommend the application of a solution of cocaine to render the tissues more bloodless; for this purpose supra-renal capsule

extract would be even more effectual. The growth is then excised by incisions well outside the limitations of the neoplasm, extending down to and including the perichondrium. Finally, the seat of the growth should be scraped with a sharp spoon. Careful examination for any enlarged lymphatic glands must be made, as when present their removal is of course essential for ultimate success. Some suitable antiseptic having been applied, the wound should be closed by suturing, as recommended by Butlin, Semon, and others, a small drainage tube being left in the lower part. The patient is fed by the rectum for the first two or three days, until he can swallow fluids through a tube lying on the side with the head lower than the body.

(3.) *Partial Laryngectomy*, the pharynx not being involved, consists in removal of the upper half or the whole of one side of the larynx, including half the thyroid cartilage. The risks of the operation, apart from relapse, are far greater than when the thyroid cartilage is left entire, and there is a growing feeling that total excision of the larynx is preferable.

(4.) *Sub-hyoid Pharyngotomy* may be adopted for the removal of growths involving the epiglottis or any epiglottic folds. Unfortunately the operation is very fatal from the peculiar liability of the patient to septic pneumonia.

(5.) *Total excision* may be undertaken with a view to employing an artificial larynx. Formerly this operation generally ended fatally from septic pneumonia, but of late the results have been improved by adopting Solis Cohen's method of detaching the larynx from the pharynx and œsophagus, drawing it forward through the median incision and dividing the trachea obliquely opposite the first or second rings. "When the larynx has been removed, the opening in the pharynx should be carefully and closely stitched up, a row of stitches being first applied to unite the edges of the mucous membrane, then a row in the muscular and soft parts around, and then the sterno-hyoid and thyroid muscles, which have been saved, will form a third layer. The skin should then be stitched up, a drainage tube being inserted at the upper angle and also through a hole well to one side, in case the wound should not remain aseptic. Finally, the divided portion of the trachea, which should be oblique from behind downwards and forwards, is stitched to the skin all round, and a tracheotomy tube is inserted into the

open orifice. In a good many cases healing is complete by the tenth or twelfth day; in some, however, where there is much tension, the wound may open and further operation may be required to close it. By operating in this way the communication between the throat and the trachea is shut off, and foul discharges cannot run down into the lungs. Further, the lymphatic infection is more thoroughly removed" (Watson Cheyne). Finally, when the pharynx is involved as well as the larynx, a more complicated operation, such as lateral pharyngotomy, with removal of the pharyngeal growth and of the whole larynx, may be available.

Palliative measures.—With the supervention of respiratory obstruction tracheotomy should be performed. Life may be prolonged for months in some cases by this operation, and in many patients there is a considerable improvement in the symptoms besides the dyspnoea. The low operation is preferable to the high, as the growth may spread down so as to render the high operation inconvenient or useless. When ulceration has occurred, the use of morphine and antiseptic applications such as aristol, orthoform, etc., is called for; or solutions of permanganate of potash, sanitas, etc., may be sprayed or gargled.

The general health and strength of the patient should of course be maintained as far as possible by the exhibition of iron, quinine, and other remedies, and constipation must be combated. The food should be soft and bland, if swallowing is painful; it is a great mistake to urge the patient to go on eating solid food when the pain and irritation of the laryngeal disease is thereby increased. With increasing difficulty in swallowing, the desirability of resorting to gastrostomy must be considered; not only does such an operation enable the patient to take more nourishment, but it obviates the constant irritation of the growth by the passage of food.

CHAPTER XI.

NEUROSES OF THE LARYNX.

INTRODUCTION—SENSORY NEUROSES—MOTOR NEUROSES.

INTRODUCTION.

Introductory remarks.—It is unnecessary to describe again in detail the intrinsic muscles and the motor nerve supply to the larynx, for which the reader will refer to pages 17 and 26. But there are certain clinical features which require emphasising. We have seen that (1,) the recurrent laryngeal nerves supply all the intrinsic muscles of the larynx, with the exception of the cricothyroid muscle, which derives its motor nerve from the superior laryngeal; (2,) both the adductors and the abductors are represented by cortical centres; and (3,) these cortical centres are bilateral in their action. It follows then that *irritation of the cortical centres of one side will result in bilateral spasm* of the vocal cords: and since the adductor and abductor centres occupy adjacent areas of the cortex at the lower end of the ascending frontal convolution, it almost invariably happens that both adductor and abductor spasm co-operate, though the action of the more powerful adductors preponderates and masks entirely the concomitant abductor irritation. Yet purely abductor spasm may apparently occur, as in the cases of hydrophobia reported by Newton Pitt.

But, on the other hand, it also follows that a *unilateral cortical lesion can never result in laryngeal paralysis*, for the bilateral action of the cortical centres of the sound side will come into play, and the laryngeal muscles will be unaffected.

So far as I know, the only two indubitable cases of bilateral cortical organic lesions of the laryngeal motor centres, or of the motor tract leading from the centres to the medulla, are those cited by Semon (*Practitioner*, Jan. 1899), viz., (a,) that of Eisenlohr, in which the post-mortem examination

revealed destruction of both optic thalami, partial degeneration in the *posterior parts of both internal capsules*, and some secondary degeneration of the pyramidal tracts in the medulla, bulbar and peripheral lesions being excluded with certainty; and (*b.*) that of Newton Pitt, in which on both sides foci of softening were found extending into the middle of the posterior part of the internal capsule. In both cases the voice was aphonic, and no sound could be uttered voluntarily, and in Eisenlohr's case laryngoscopic examination (which was impossible in Pitt's case during the aphonic stage) revealed bilateral adductor paralysis. But in both cases respiratory difficulties were conspicuous by their absence, corresponding to the fact that the physiological act of respiration is mainly represented in the medulla. Semon draws attention to the correspondence between the lesions in these cases and the results of Horsley's and his own demonstration of the position of the phonatory tract in the internal capsule.

It has been demonstrated by Semon that "there exists an actual difference in the *biological* composition of the laryngeal muscles and nerve endings," rendering the abductors more prone to be affected by conditions resulting in paresis and atrophy than the adductors, "whilst the fact that also in central (bulbar) organic affections, such as tabes, the cell groups of the abductors succumb earlier than those of the adductors, points to the probability that similar differentiations exist in the nerve nuclei themselves." Thus, in all progressive organic lesions of the centres or trunks of the motor laryngeal nerves, we find that the abductor muscles alone are first paretic or paralysed, and that complete paralysis from subsequent involvement of the adductors supervenes after a long or short interval, unless, of course, the lesion is so gross as to produce a total and complete motor paralysis of the laryngeal nerves *ab initio*. This increased vulnerability of the abductors, as compared with the adductors, is known as "Semon's law." The order then in which the muscles succumb is first the crico-arytenoidei postici, then the thyro-arytenoidei interni, and finally the crico-arytenoidei laterales; and in recovery from lesions producing paralysis the converse order is observed.

Thus, not only are the *postici* muscles more prone to succumb to organic disease than the *laterales* (or adductors), but the adductor nerve fibres also tend to regenerate and recover functional activity sooner than the abductor fibres.

A remarkable proof of this is afforded by the case of A. P., a boy aged 15, under the care of one of my surgical colleagues, at the Bristol Royal Infirmary. In performing an operation for septic thrombosis in the right internal jugular vein, under conditions which rendered the procedure especially difficult, the right vagus nerve was divided below the superior laryngeal

branch, the cut ends being sutured immediately. When I examined the larynx shortly afterwards, the right cord was completely paralysed, and it remained in this condition for several months. But on examining the larynx in July, 1894, seven months after the operation, the right cord was in the middle line, and the boy had an excellent and powerful voice. Thus there was no longer complete paralysis, but only abductor paralysis. The patient was seen by Sir Felix Semon and Dr. McBride, both pronouncing it to be a case of simple abductor paralysis of the right cord. A similar case is recorded by Makins.

It may be mentioned that the views of Semon in explanation of the median position of the vocal cord in the earlier stages of progressive vocal cord paralysis are not universally accepted as yet. Moreover, other hypotheses have been advanced. Thus, Mackenzie considered that it might be due to the abductor nerve filaments being placed most superficially in the nerve trunks, and therefore the first to succumb; Sir William Gowers, that it was a consequence of the greater mechanical advantage at which the chief adductor muscle was placed in its attachment to the muscular process of the arytenoid cartilage, as compared with the abductor muscle; Grützner, that the adductors might be regarded as a "white" muscle, the abductor a red muscle, and that this accounted for their difference in vulnerability; Grabower that the nerve endings of the abductors, being more complicated than those of the adductors, renders them more vulnerable (see *Plate VII*). Krause till recently attributed the median position first assumed by the vocal cord to a primary neuropathic contracture of all the muscles supplied by the recurrent nerve, with resulting preponderance of the adductor; in other words, the median position was attributed to prolonged spasm of the glottic muscles. It is, however, inconceivable that there should exist a spasm of the adductors persisting over periods of many months, and in some cases even as long as twelve years; and moreover, as Semon has pointed out, it is impossible to accept such a theory which leaves quite unexplained the commonly observed fact, that the postici muscles have often almost completely atrophied in the more chronic cases, while the laterales remain normal in size and appearance. Similar objections apply to Wagner's theory, that the median position of the cords is due to an adducting action by the crico-thyroid muscle.

The paralysis of the internal thyro-arytenoidei, which is often associated with the paralysis of the abductors due to chronic organic lesions, is a further proof that the condition is one of paralysis, and cannot be of the nature of a primary contracture.

One other hotly debated question of clinical importance must be briefly referred to, viz., "What is the position assumed by the vocal cord in complete paralysis of all the muscles supplied by the recurrent nerve?" Many observations have been made on this point, and several experimenters, *e.g.*, Semon, Horsley, Traube, Klemperer, Schech, and others, whilst directly inspecting the vocal cords during section of the recurrenents, have seen the corresponding vocal cord at once assume the cadaveric position immediately upon section of the nerve. The cadaveric position, then, is the position of complete recurrent nerve paralysis in man, and not the median position, as claimed by Grossmann from experimental observations on the cat.

SENSORY NEUROSES.

The sensory neuroses of the larynx may be conveniently grouped under two divisions: (*a*.) Anæsthesia; (*b*.) Hyperæsthesia and Paræsthesia.

ANÆSTHESIA.

Etiology and Pathology.—The loss of sensation may extend to the whole of the larynx and upper part of the trachea, or it may be less extensive and confined to the epiglottis, or to one side of the larynx, or to the supra-glottic portion. Again, it may vary in degree from slight diminution to complete absence of sensation.

The anæsthesia may be due to *peripheral* lesions, *e.g.*, diphtheria, syphilis, injury to the vagus or superior laryngeal nerves, or it may be *central* in origin, *e.g.*, bulbar lesions, locomotor ataxia, general paralysis of the insane, apoplexy, epilepsy; and it often occurs, though usually in a minor degree, in hysteria. Apart from the fact that the superior laryngeal nerve is also the motor nerve to the crico-thyroid muscle, when we consider the nature of the lesions which cause anæsthesia, it obviously must generally be associated with motor paralyses of the laryngeal muscles, and, in many cases, with lesions of other cranial nerves.

Symptoms.—The symptoms consist mainly in a tendency for food to enter the larynx and produce spasm and choking attacks. When anæsthesia is complete and subglottic, spasm of the larynx does not occur, and the food particles then enter the lower air-passages and may either cause violent obstructive dyspnoea or, becoming impacted in the smaller bronchi, may set up pneumonia. Thus the prognosis is very grave if the lesion is bilateral.

Mucus and small particles of food tend to collect in the supraglottic portion of the larynx, and if the anæsthesia be unilateral the tendency is confined to the affected side.

The *diagnosis* can only be made by probing the laryngeal mucous membrane, when the defect or absence of sensation is readily detected. It is important to note any co-existing paresis of the laryngeal or pharyngeal muscles, or anæsthesia of the base of the tongue, palate, etc., and that any further evidence of central nerve-disease should not be overlooked.

Treatment consists in special care in feeding the patient. If bilateral, food should be given by the œsophageal tube, and, in passing the tube, it must be remembered that the usual indications of its having entered the glottis cannot be manifested, inasmuch as the larynx is insensitive. If the patient be made to utter a sound after the tube has been passed, proof is afforded that it has not been passed into the trachea; for with the tube between the vocal cords, it is impossible to phonate. When the anæsthesia is due to diphtheria, local faradisation and the administration of strychnine should be resorted to, and of course in syphilis of the central nervous system, iodide of potassium or mercurial preparations will be given.

HYPERÆSTHESIA AND PARÆSTHESIA.

Pain may be due to local disease in the larynx, but in such cases it is merely a symptom of the organic lesion, and is not comprised in the term "neurosis." A variety of sensations, described as rawness, constriction, pain, pressure, or tickling, the sensation of a pin pricking, or of a fish-bone in the throat, are met with in anæmic, hysterical, or hypochondriacal patients. It is well to remember the fact, to which attention has been already directed elsewhere, that the power of localising sensations felt in the throat is very defective, and that all sensations arising from any part of the throat are subjectively referred to the laryngo-tracheal region. Sometimes such symptoms result from reflex irritation, due to enlarged faucial or lingual tonsils, but usually physical examination reveals no local cause for the sensations. The purely neurotic cases are commonly associated with somewhat similar pharyngeal symptoms.

The *diagnosis* depends on the exclusion of any organic lesion, but will generally rest mainly on the general symptoms of the neurotic temperament. Special care must be observed to exclude tuberculous disease, as perverted sensation in the larynx is not infrequently one of the earliest manifestations of commencing tubercle of the lung. Hyperæsthesia is sometimes a marked feature in gouty or rheumatic affections of the respiratory tract, and likewise in locomotor ataxia. The general history of the case should lead to the detection of the former diatheses, while the co-existence of laryngeal crises, with persistent frequency of the pulse and abductor paresis, would confirm the suspicion of the latter disease.

The **treatment** consists in the exhibition of nervine tonics and improvement of the general health by dieting, baths, a sea voyage, etc. When associated with uterine disorder, and especially in patients at the climacteric, appropriate general treatment will often afford relief; but in a considerable number of cases treatment of any kind is very disappointing, and any local treatment of the larynx rarely desirable. When the symptoms definitely appear to result from diseased tonsils, or other removable causes, they should, of course, receive attention.

MOTOR NEUROSES.

HYPERKINESIA—SPASMODIC AFFECTIONS.

Spasmodic affections of the larynx may be grouped under three divisions: (*a*,) Respiratory spasm: (*b*,) Phonatory spasm; (*c*,) Neuroses of co-ordination.

Glottic spasm may arise from irritation in any portion of the nerve supply to the larynx, either the nerve centres, or nerve trunks.

Glottic spasm is usually due to reflex or other causes acting on the nerve centres, and is therefore bilateral, but in children, owing to the small size of the glottis, spasm of one cord may produce sufficient narrowing to cause urgent dyspnœa. In the wider adult larynx, although glottic spasm may result from irritation of one vagus nerve, it is never due to pressure on one recurrent nerve. Just as stimulation of the peripheral end of a divided recurrent laryngeal nerve causes adduction of the corresponding cord, although both the abductor and the adductor fibres are equally stimulated, so in glottic spasm the adductors preponderate, with resulting closure of the glottis, although probably both abductor and adductor spasm really occurs.

LARYNGISMUS STRIDULUS.

Etiology.—The affection *Laryngismus Stridulus* (or "*false croup*"*), is almost invariably associated with rickets, or in ill-nourished, badly-developed, or hydrocephalic children; it is also observed in tetany. It may occur at any age up to the seventh or eighth year, but is most frequent between the ages of six months and two years.

* The term "*false croup*," if ever employed, should be reserved for acute laryngitis in children.

The remarkable excitability of the nerve centres accompanying rickety conditions is the usual predisposing cause, but the spasms are generally directly excited by irritation, either in the alimentary tract, from undigested food or from parasites, or it may be due to such causes as post-nasal growths, a pendulous epiglottis, or enlarged bronchial glands.

Symptoms.—The attack consists in a few short stridulous inspirations, followed by sudden spasmodic closure of the glottis, with absolute cessation of respiration, lasting ten or fifteen seconds, followed by a long crowing inspiration, either continuous or interrupted, as in sobbing. The child stops playing and stands with staring eyes, the pupils contracted, the head thrown back, the face at first pale, then livid with increasing asphyxia, and bedewed with clammy perspiration till the attack ceases. After the attack is over the child may be tired or irritable, or may fall asleep. In other cases it will go on with whatever it happened to be doing as though nothing had occurred. The muscular spasm may extend to the facial muscles, or the thumbs may be turned in, and carpo-pedal convulsions, or even general convulsions may supervene.

The attacks vary much in severity, and the first may be fatal from the persistence of the spasm and asphyxia, or the glottis may remain closed till unconsciousness occurs. The prognosis is especially grave in these silent cases, and in those in which the spasm is so marked that the inspiration is interrupted and sobbing. In the less severe forms the carpo-pedal spasms may be absent and the symptoms less definite, and parents sometimes speak of the attacks as "passion fits," or "holding the breath."

The attacks may occur very occasionally, or there may be several daily.

The **Diagnosis** may be made from the character of the attacks, with sudden onset and complete cessation of respiration, and with freedom from symptoms which indicate laryngeal disease or growths in the intervals, such as cough, hoarseness. Congenital laryngeal stridor may be mistaken for laryngismus stridulus; but in the former there are no carpo-pedal contractions, the stridor is peculiar, more persistent, and dates from within a few days of birth, and is not sudden in onset (see p. 179).

Prognosis.—In severe cases the prognosis should be guarded, for death from asphyxia has occurred in a large number of

cases, and especially is this liable to happen in the silent cases. Nevertheless, the affection is generally amenable to appropriate treatment, and only a small percentage terminate fatally.

Treatment comprises in the first place the cure of rickets or other general conditions that may be present, the removal of fecal accumulations, parasites, etc. Warm clothing, fresh air, and simple diet, and avoidance of mental excitement, are of first importance, while as regards drugs in rickets, nothing is so valuable, in my opinion, as minute doses of phosphorus.

As a rule, the attack passes off spontaneously in a few seconds, but if persistent and if asphyxia is threatened, the best plan is to dash cold water in the face, while the legs and body may be immersed in a hot bath. The spasm can sometimes be relieved by hooking forward the epiglottis with the forefinger.

If the attacks recur frequently, small doses of nitro-glycerine, bromide of potassium, belladonna or chloral, will tend to keep them off and render them less severe.

GLOTTIC SPASM IN ADULTS.

Etiology.—Spasm of the sphincters of the glottis in adults may be due to (1,) peripheral reflex, (2,) central functional, or (3,) central organic affections.

Most commonly it is a *reflex* phenomenon dependent on morbid conditions of the larynx, e.g., the presence of a neoplasm, tubercular disease, catarrhal conditions, or it is due to irritation of the motor nerves by pressure of aneurysms, growths, etc. It is sometimes associated with adenoid hypertrophy of the tongue, elongated uvula, or with gouty or rheumatic laryngitis.

Paroxysmal glottic spasm, associated with a peculiar brassy cough, may be one of the earliest indications of intra-thoracic aneurysm or mediastinal growth. The possibility of some such grave organic lesion should always be borne in mind. [See **Aortic Aneurysm.**]

Functional inspiratory spasm, the so-called hysterical spasm, is not usually severe, yet, in some instances, the closure of the glottis may be prolonged, and unconsciousness or frequent shallow respirations, with incomplete closure of the glottis, may persist for several hours or days, the attacks being very liable to recur; from slight local causes—as, for instance, a laryngoscopic examination—or from emotional disturbance.

There may be defective abduction during voluntary inspiratory efforts resembling abductor paralysis, though in reality an inspiratory spasm. But if the patient is directed to phonate as long as he can without stopping, he soon makes a deep inspiration involuntarily, and the cords are then abducted by the reflex action of the bulbar centre. Functional inspiratory spasm may be associated with spasm of the soft palate, as in a case shown by Willis at the London Laryngological Society. Hysterical paralysis of the pharyngeal muscles or the œsophagus is occasionally met with, and may cause dysphagia.

A remarkable instance of this affection is recorded by Hopkins, which commenced with pertussis, but persisted, the ordinary whoop being eventually replaced by a high-pitched, piercing squeal, like that of a badly hurt pig. At first inspiratory, the squeal became both expiratory and inspiratory, the attacks being preceded by a tickling in the throat. The larynx was normal, no other hysterical stigmata were present; but no treatment afforded more than temporary relief until the larynx was intubated, and then, although the tube was only retained for less than an hour, the cure appeared lasting and complete.

Glottic spasm occurs also in certain lesions of the nerve centres, *e.g.*, the laryngeal crises of tabes dorsalis, in hydrophobia, diphtheria, and in tetany.

Treatment.—The exciting cause, especially any indications of intra-thoracic aneurysms or new growths, should be ascertained, and, where possible, receive appropriate treatment. In functional cases general dietetic and hygienic regulations are mainly called for. In the laryngeal crises of locomotor ataxia the inhalation of amyl nitrite gives relief, and sometimes the attacks, if slight, may be warded off by cocaine sprayed into the larynx, while other measures appropriate to the treatment of "crises" generally are demanded. In these cases there seems to be an abnormal excitability of the abductor centres; it is therefore desirable to give directions to the patient to avoid as far as possible, all sources of irritation in the larynx, *e.g.*, smoking, drinking cold fluids, etc.

Locally the application of the faradic or interrupted galvanic current, or of stimulating inhalations, may be tried.

Bidon has found digital compression of the phrenic nerve between the two inferior attachments of the sterno-mastoid efficacious in arresting laryngeal spasm. The method was introduced by Leloir as a means of arresting nervous hiccough.

NERVOUS LARYNGEAL COUGH.

This is a condition in which the glottis is spasmodically closed, and followed by a short, loud, harsh, barking cough, termed by Sir Andrew Clark the "barking cough of puberty." It occurs in young persons of both sexes. The cough, which usually ceases during sleep, generally occurs once at a time, not a series of successive coughs (thus differing from the cough due to sensory laryngeal irritation), and recurs persistently, and more or less rhythmically, throughout the day, even during rest. It is sudden in onset, is unattended with constitutional symptoms, abnormal physical signs in the chest, or expectoration, and generally stops when the patient's attention is distracted. The voice is not in any way impaired, and there is no shortness of breath involving forcible inspiration after the cough. In fact it is simply a sudden closure of the glottis with a forcible expiration, due to involvement of both the laryngeal and respiratory branches of the vagus.

This affection is really one of the "convulsive ties," and not in any way associated with volitional acts. Only occasionally are general choreiform movements present in this affection, which has been unfortunately termed "laryngeal chorea." In every case a particular group of muscles is involved, thus differing from the irregular movements of true chorea. It should be carefully distinguished from the spasmodic cough due to irritation of the vagus or its branches by an aneurysm, growth, or enlarged bronchial gland, and the greatest caution should be observed in the less typical and doubtful cases in excluding slight ulceration in the larynx, or the earlier manifestations of lung disease.

Spasmodic Laryngeal cry, persistent Hiccough, etc. — The "hydrocephalic cry," described by Trousseau as characteristic of cerebral meningitis, though now known to occur in other affections, is probably due to irritation of the cerebral cortex. Very rarely a somewhat similar "cry" constitutes the main feature of cortical irritability, as in the case of a boy aged eleven, brought before the London Laryngological Society by Bond. For eighteen months, with one free interval of three months, he had continued to utter a loud, sudden, explosive cry of considerable volume, very like part of a milkman's cry, at irregular

intervals varying from about twelve seconds to one and a half minutes. The cry was associated with somewhat violent action of the diaphragm, and with a lifting of the soft palate, but though for a long period it would continue to be uttered during sleep, it was never emitted during laryngoscopic examination. The boy seemed dull and stupid; his hands and arms were continually working somewhat like those of a child with chorea; he had adenoids and double proptosis. Bond considered that it was a case of "tic convulsif."

Persistently recurring hiccough, a condition analogous to nervous laryngeal cough and spasmodic laryngeal cry, though not a laryngeal affection, may be mentioned in connection with these diseases.

The **Treatment** appropriate to these conditions consists in attention to the general health, diet, and abundance of fresh air, regular exercises, baths, etc. The most useful drugs are iron, arsenic, and strychnine, and for ill-nourished children, cod liver oil and malt extract. In the case of young females, the "barking cough of puberty" may be an indication for the exhibition of such uterine tonics as pulsatilla and caulophyllum. Though, of course, any obvious disease, such as adenoid growths, should be removed when present, local laryngeal treatment is seldom desirable: yet occasionally, in cases of long standing, the use of the faradic or galvanic current may prove useful, as in a case of nervous laryngeal cough of two years' standing, in which Bond obtained a cure in a few minutes by faradism.

PHONIC SPASM.

Phonic Spasm (*Dysphonia spastica*, Schnitzler) is a rare affection in which spasm of the adductors and tensors of the vocal cords occurs only on attempted phonation. It is essentially an occupational neurosis, analogous to writers' cramp, with which it has been observed to co-exist. B. Fränkel's cases of *mogi-phonia*, in which spasm, with impairment or loss of voice, occurs on singing or attempts at public speaking, and in which ordinary conversation is not interfered with, come under this head.

Though very rarely met with in other persons, the patients are nearly always professional voice users of a nervous temperament, and the condition first manifests itself by weakness or loss of voice coming on shortly after commencing to read, preach, or

sing, as the case may be. As the condition progresses, using the voice in a professional capacity is at once attended with spasmodic closure of the vocal cords, the endeavours of the patient to force a current of air through the glottis being futile as long as he attempts to speak, but ceasing as soon as he desists. "The patient is often able to produce some notes, either in his own natural voice or in a slightly muffled tone; but while he is speaking in this way, the current of the voice seems to be partially interrupted, and the sound conveys the idea of an arrested action of the respiratory muscles" (Mackenzie). During the attempt to phonate, the vocal cords may be seen to be so forcibly adducted that no chink is left for the expiratory current of air. The affection constitutes one form of stammering (see *Stammering*, below).

In connection with *mogiphonia* we may allude to two other rare neuroses, viz., *apsithyria* (Solis Cohen), or inability to whisper, and *hysterical mutism* (Charcot), which similarly depend on psychical functional disturbance. But as these are functional paralyses, not spasmodic affections, they will be referred to in a later chapter (page 269).

Treatment is generally very disappointing, but rest, abstention for a time from the occupations with which the affection is associated, nervine tonics and the treatment of any local disease of the upper respiratory tract are the main indications. Any faulty method of producing the voice should be noted and corrected.

ATAXIA OF THE VOCAL CORDS.

Irregular, disorderly, choreic movements of the cords have been observed accompanying general chorea; and in various diseases of the central nervous system, the larynx may be involved, and abnormal movements of the vocal cords may result. Thus, for instance, in disseminated spinal sclerosis a tremor of one or both cords may occur on phonation.

STAMMERING, STUTTERING.

Under the term stammering in its wider sense it has been customary to include not only stuttering, a spasmodic affection of the organs concerned in the mechanism of speech, causing the enunciation of words or syllables to be checked,

but also many defects in articulation, such as the inability to pronounce certain combinations of letters, lisping, the interjection of meaningless sounds in the pauses between words, conditions which will not require consideration here.

In the mechanism of speech there are two factors :—

(a,) *Vocal*, comprising two elements, viz. :—

(1,) The *laryngeal*, phonation depending on adduction of the vocal cords.

(2,) The *respiratory*, expiration through the partly-closed glottis, depending on the diaphragm and other muscles of expiration.

(b,) *Oral*, the modification of the vowel sound by the tongue, lips, teeth, or soft palate in sounding consonants.

In normal speech these two factors are perfectly co-ordinated; in stammering there is imperfect co-ordination due to irregular muscular action in either the oral, laryngeal or respiratory element.

Oral Stammering.—Normal speech has been aptly compared by Wyllie to playing the violin, in which one hand draws the bow by which the musical sound is produced, while the other modifies this initial sound by “fingering.” Simply pressing the fingers on the violin string will not give a series of notes; it is essential that the bow hand should co-operate by producing the musical note which the “fingering” can modify. So in the mechanism of speech there are two factors—the *vocal* and the *oral*, the former corresponding to “bowing” the violin string, the latter to “fingering.”

Though, as we shall see, stammering may be “laryngeal” or “respiratory,” in the great majority of cases it is due to imperfect co-ordination of these vocal and oral elements, the vocal element being delayed. Take for example the word “cap.” The initial “c” cannot be sounded except in conjunction with the vowel sound, but the stammerer’s vowel sound is delayed, consequently the attempt to sound the “c” results only in characteristic guttural sounds, or no sound may be emitted. The difficulty appears to him to be in sounding the “c,” and so he makes repeated and more forcible efforts to get it out, until at length the vowel sound too comes out with undue energy, thus, “ck-ck-ckap.” He gets the impression that the stammering is due to a difficulty in pronouncing the consonants, and thus greater attention and stress is given to the consonants,

with forcible or spasmodic overaction of the muscles, and the stammering becomes habitual; yet the difficulty really lies in the failure to produce the vowel sound in the larynx. If the stammerer's attention be diverted from the consonant to the vowel sound, the difficulty disappears, and thus there is often no hesitation in pronouncing words commencing with a vowel sound, inasmuch as the *vocal* mechanism is called into action first, and the vocal sound once started is not interrupted by the interjected consonant. For instance, a stammerer who stammers over "cap," will often not do so over "apple." When, however, two or more consonant sounds occur together, requiring rapidly varied co-ordinated movements of the tongue or lips, as in the words "*abduct*," "*astride*," he will be more likely to break down.

Conversely in most cases stammering does not occur during singing or intoning which involves continuous phonation, and in which the vowel sounds predominate.

Pharyngeal stammer is associated with irregular contractions of the muscles of the palate.

Laryngeal stammer is due to spasmodic closure of the glottis, and *respiratory stammer* to imperfect action of the muscles of expiration, especially the diaphragm.

The degree of stammering varies largely in different cases. In the milder forms the stammer is only present when the sufferer is nervous or self-conscious, or only affects certain consonants, especially the explosive consonants, or it may be only when two or more explosive consonants occur together. But in the severer forms the nervous energy expended on the motor cortical speech centre irradiates and involves other motor areas, with convulsive movements of the face, limb and trunk muscles. In such a case, if, for example, the spasm is associated with a guttural consonant and begins at the back of the tongue, the mouth remains open, the muscles of expression twitch, the veins of the face and neck are distended (for expiration is impossible while the spasm lasts), the arms and legs jerk or are violently convulsed, and the sufferer's aspect is truly pitiable. All gradations between the mild and graver forms occur. In some cases, voluntary movements of the limbs seem to assist the speech, thus one of my patients found that standing and moving one leg made it less difficult to overcome the stammering.

Etiology.—Stammering is sometimes hereditary, and like other neuroses is especially prone to occur in those of the nervous temperament. It is seldom observed before the fifth year, and though usually commencing before the period of puberty is passed, it may begin in later life. It may follow some exhausting illness, or be due to a nervous shock or a fright; in other cases it is started by imitation, and becomes a confirmed habit.

Formerly it was generally held that stammering was due to structural defects or abnormalities of the speech mechanism, but since Arnott suggested that it was caused by delayed action in the vocal mechanism, that is to say, in phonation, this view has been generally accepted, and is essentially correct.

In a large proportion of stammerers no defect will be found in the upper respiratory tract, but in others various abnormal conditions are present, such as enlarged tonsils, post-nasal growths, chronic pharyngitis, lingual adenoids, etc. In some cases, no doubt, these co-existing defects have had no influence in causing the stammering, and in others the abnormal muscular speech efforts have caused the local disease; but, like Bryson Delavan, I am convinced that in a few cases the habit largely owes its inception to diseased conditions in the pharynx, and that it is highly desirable to remove these physical defects before attempting, by appropriate exercises, to treat the stammering.

Treatment.—Having satisfied oneself that there is no co-existing disease of the upper respiratory tract which ought to be rectified, the treatment consists *firstly*, in improving the general health and physique, by open-air exercise and games, etc., and the avoidance of any mental overwork; and, *secondly*, by educational methods to acquire perfect co-ordination of the speech mechanism; *thirdly*, in the rectification of abnormal respirations during phonation by means of breathing gymnastics (Coen): especially (1,) deep continued inspiration; (2,) short expiratory movements; (3,) gradually prolonged expiration; (4,) holding the breath.

With patience and perseverance both on the part of the patient and his instructor, there are very few cases that cannot be cured, or at least so greatly improved that the defect is hardly noticeable; but it is beyond the scope of this work to give the details of the exercises and methods to be followed.

LARYNGEAL VERTIGO.

(ICTUS LARYNGIS.)

Charcot, in 1876, directed attention to a condition which he named "laryngeal vertigo," characterised by a tickling sensation in the throat giving rise to coughing, followed by spasm of the glottis and momentary loss of consciousness, which is completely recovered from in a few seconds, and is not usually followed by stupor or any indications of the patient having had an epileptic fit. The term "vertigo" is unfortunate, inasmuch as in most of these cases there is no true vertigo. McBride explains the phenomena of laryngeal vertigo by the action of forced expiration with a closed glottis, since Weber showed that a somewhat similar condition of loss of consciousness may be induced by voluntary forced expiration with a closed glottis.

Doubtless an essential predisposing cause is an unstable nervous system, the glottic spasm and cough being often excited by some abnormality in the respiratory tract. The actual cause of unconsciousness probably differs, in some being truly of an epileptic character (*petit mal*), in others due to syncope. In one case under my care the attacks were always induced by compression of the larynx.

Getschell, who collected reports of forty-one cases, states that the average age of the patients was against the theory of its being epileptic in origin, but he is equally opposed to the "forced expiration" theory, and he considers that the circulatory condition is nothing more than an exciting cause in a limited number of cases. An analysis of the forty-one cases shows that sixteen were over fifty, thirteen between the ages of forty and fifty, and all the others were younger. One was epileptic, and one had an epileptic brother. Loss of consciousness was reported in thirty-two cases, and falls in twenty-six. True vertigo was mentioned in one case only, in five slight mental confusion was noted, and dizziness in eight. Cough preceded the attacks in thirty-three cases, and bronchitis occurred in eight cases.

Treatment.—If associated with a catarrhal condition of the respiratory tract, this requires treatment. In many cases the patient is otherwise healthy, and all that can be done is to administer such remedies as the bromides, and attend to the general health and hygienic conditions. It is sometimes

associated with gouty manifestations. Digital compression of the phrenic nerve, as recommended by Bidon for glottic spasm, might be tried in laryngeal vertigo. Merklen has recorded two cases cured by the administration of antipyrin in daily doses of 20 to 40 grains. The author's patient was always relieved by applying hot water to the laryngo-tracheal region.

LARYNGEAL PARALYSIS.

Motor palsy or paresis of the laryngeal muscles may be due to lesions in any portion of the motor nerve tract from the cortex to the termination of the nerve fibres in the muscles. The source of the paralysis may therefore be: (1,) In the cerebral cortex, or internal capsule. Since the lesion must involve the centres of both hemispheres, cortical laryngeal palsy is almost invariably a functional neurosis; (2,) In the medulla oblongata—either due to degeneration of the vago-accessory nuclei—or to pressure by a growth, or to meningeal inflammatory thickening; (3,) Pressure on or destruction of the motor nerve fibres in the vagus or its recurrent branch, either by intracranial growths, or growths in the neighbourhood of the jugular foramen at the base of the cranium or in the neck or thoracic cavity; (4,) In the muscles themselves, the so-called myo-pathic paralyses.

PARALYSIS OF MUSCLES SUPPLIED BY THE SUPERIOR LARYNGEAL NERVE.

PARALYSIS OF THE CRICO-THYROID MUSCLES.

Etiology.—The function of the only muscle supplied by the superior laryngeal nerve, viz., the crico-thyroid, is to assist in rendering tense the corresponding vocal cord on phonation. Isolated paralysis of this muscle is rare, but it naturally occurs in association with the anæsthesia of the laryngeal mucous membrane due to section of the superior laryngeal nerve, pressure of enlarged glands or to its involvement in new growths, etc. It is said to have been caused by cold or overstraining of the voice, and by diphtheria.

Symptoms.—*Total paralysis* is attended by anæsthesia of the larynx, the symptoms of which have already been referred to. The laryngeal appearance in bilateral paralysis of the superior laryngeal nerves is not very characteristic. The vocal cord

presents a wavy outline, and owing to defective tension it bulges upwards in the centre on forced expiration, and is depressed on inspiration. When the paralysis is unilateral, this is not difficult to recognise, but in the very rare bilateral cases these features would be less obvious. If unilateral the affected cord appears higher than the sound one, during phonation.



FIG. 119.
Unilateral paralysis of
the superior laryngeal
nerve. (Phonation.)



FIG. 120.
Bilateral paralysis of
the superior laryngeal
nerve. (Phonation.)

The treatment of the anaesthesia of the larynx has already been alluded to; for isolated paralysis of the crico-thyroid, external faradisation or counter irritation, and internally the exhibition of strychnine may be recommended.

PARALYSIS OF THE MUSCLES SUPPLIED BY THE RECURRENT LARYNGEAL NERVE.

In the introductory remarks at the commencement of this chapter it has been shown that in all progressive organic lesions involving the motor nerves to the larynx or their bulbar nuclei of origin, the abductor fibres are the first to succumb, then those to the internal tensors of the vocal cords, and finally, with complete recurrent paralysis, the adductors. It is probable that in all cases due to organic disease, with the exception of those resulting from injury to, or section of a recurrent nerve, which of course at once produces complete recurrent paralysis, the abductors are first paralysed for a longer or shorter period; although, as we shall see, the abductor paralysis when unilateral rarely gives rise to symptoms which attract the attention of the patient or physician, and therefore is doubtless often overlooked until, with the supervention of complete paralysis, the voice becomes affected. But in some affections only the adductors may be

paralysed, and in others again the inter-arytenoideus is alone involved. For clinical convenience, therefore, paralysis of the muscles supplied by the recurrent laryngeal nerve will be described in the following order : (*a*,) Paralysis of the adductors of the vocal cords ; (*b*,) Paralysis of the abductors of the vocal cords (posticus paralysis) ; (*c*,) Paralysis of internal tensors of the vocal cords ; (*d*,) Complete recurrent paralysis ; (*e*,) Paralysis of the inter-arytenoideus muscle.

PARALYSIS OF THE ADDUCTORS OF THE VOCAL CORDS.

Adduction of the cords is brought about by the action of the crico-arytenoidei laterales arising from the sides of the cricoid cartilage, and passing backwards and upwards to the external angles of the arytenoid cartilages. Their contraction causes inward rotation of the arytenoids on their axes, causing the vocal cords to approach in the middle line. But for perfect adduction of the cords the arytenoids must also be approximated by the arytenoideus and thyro-arytenoidei externi muscles. If the arytenoideus be paralysed alone, a triangular chink is left on phonation behind the closed vocal cords.

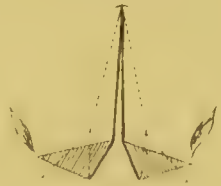


FIG. 121.

To illustrate the action of the crico-arytenoidei laterales in adducting the vocal cords.

Unilateral Paralysis of the Adductors alone is extremely rare, and can only be due to local causes or to toxic paralysis. It is reported to have occurred from cold, syphilis, enteric and typhus fever, small-pox, etc. Some cases have been reported by Stewart, ascribed to reflex influences from the nose. Morell Mackenzie observed unilateral adductor paralysis in a case of lead poisoning.

The appearance presented by the larynx is not very characteristic, and is liable to be mistaken for complete paralysis of one vocal cord. The examination should extend not only to observation of the larynx during phonation and quiet respiration, but also during deep inspiration when, unless the adductor paralysis was complete, further abduction would take place. In complete adductor paralysis the vocal cord would be completely abducted, showing a concave margin, and, the healthy cord being hardly able to pass across to meet it perfectly, the voice must be weak.

Bilateral Adductor Paralysis is generally functional, and due to hysteria, or to various reflex causes, *e.g.*, uterine. But it may also be due to general weakness, anæmia, or to catarrhal affections of the larynx. The vocal cords, on attempted phonation, either remain divergent, as in deep inspiration, or are imperfectly approximated. In hysterical cases the voice is lost but the cough, and sometimes the laugh, is phonic, while in the very rare cases due to local lesions, cough is aphonic, or, rather, is altogether impossible.



FIG. 122.

Bilateral adductor paralysis during vocalisation.

The paralysis is rarely complete, and usually amounts to paresis only. In hysterical cases the onset, and often the recovery, is sudden, but when due to general weakness or to local catarrhal conditions with infiltration of the muscles, the onset, like recovery, is naturally more gradual.

Functional adductor paralysis can often be cured by internal faradisation, one pole of the battery being placed externally over the episternal notch, the other being formed by an intralaryngeal electrode which is guided into the larynx by the laryngoscopic mirror. The circuit is completed as soon as the electrode reaches the glottis: if the current be strong enough to be slightly painful, the patient usually makes an ejaculation, and on withdrawing the electrode the voice is restored. It is better to apply a fairly strong current at once instead of trying very feeble currents, since the moral effect produced has a good deal to do with the success of the procedure. The exhibition of various preparations of iron and other tonics is called for in many cases, and in some patients in whom the intralaryngeal faradic current is unsuccessful, these general measures alone may have the result desired. (See also "Hysterical mutism" and "Apsithyria," p. 269).

PARALYSIS OF THE ABDUCTORS OF THE VOCAL CORDS.

The vocal cords are abducted by the crico-arytenoidei postici muscles which, arising from the posterior surface of the cricoid cartilage, pass upwards and outwards to be attached to the external angles of the arytenoid cartilages. By their contraction the arytenoids are rotated outwards on their axes, and the vocal cords are carried outwards, *i.e.*, abducted. These muscles are used in deep inspiration.

Etiology.—The most common cause is pressure on the recurrent laryngeal, either by an aneurysm, tumour, goitre, enlarged mediastinal glands, tubercular thickening of the right apex of the lung, or by malignant disease or a foreign body in the œsophagus. It frequently results from central nerve lesions in the medulla, or to implication of the vagus or spinal accessory nerves at the base of the brain, *e.g.*, in locomotor ataxia, disseminated cerebro-spinal sclerosis, bulbar paralysis, hæmorrhages in the medulla, apoplexy, syphilitic nuclear disease, or thickening of the dura mater.

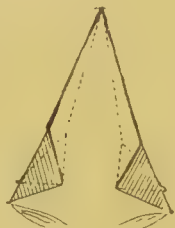


FIG. 123.

To illustrate the action of the crico-arytenoidei posticus muscles in abducting the vocal cords.

It may be due to *toxic neuritis*, from pneumonia, typhoid or typhus fever, diphtheria, scarlet fever, rheumatism, influenza, lead, arsenic, alcohol; or may be *myopathic* in progressive muscular atrophy, or wasting diseases. Heymann collected over fifty reported cases of toxic paralysis of the larynx, and he found that lead was by far the most common cause, and usually mainly affected the abductors. Toxic paralysis has been attributed to copper, phosphorus, morphine, antimony, atropine and cannabis indica, but I know of no instances recorded which are not open to doubt.

Unilateral Paralysis of the Abductor may be due to any of the above causes, but is most frequently met with as the result of pressure on, or implication of, a recurrent nerve. The reason why abductor paralysis generally occurs before complete abductor and adductor paralysis supervenes has already been discussed.

The affected cord remains in the middle line on deep inspiration, being persistently retained in the position of adduction by paralytic contracture of the adductor muscle. Consequently, as phonation is not interfered with, and respiration is seldom much embarrassed except on exertion, the condition is frequently overlooked.

Bilateral Abductor Paralysis is less common. It may be due to cold, diphtheria, or to any of the causes producing the unilateral affection.

The cords are kept in the median position by the gradually developed paralytic contracture of the adductors, and only a small chink is seen between them. They do not abduct at all

on inspiration if the paralysis is complete. The diagnosis may be made by directing the patient to phonate for some seconds during the inspection of the larynx, and then take a deep inspiration, when the vocal cords instead of abducting are drawn together. Consequently respiration is greatly embarrassed if the



FIG. 124.

Bilateral abductor paralysis in a case of locomotor ataxia on deep inspiration.

paralysis is complete, but abductor paralysis is fortunately often incomplete. Paroxysmal attacks of urgent dyspnoea with stridulous or sonorous inspiration are liable to occur on slight exertion or mental excitement and even during sleep, and may at any time end in sudden and fatal asphyxia. But the voice is unaltered.

The prognosis of bilateral abductor paralysis is therefore generally exceedingly grave, and at any moment tracheotomy may be urgently demanded. In progressive lesions the thyro-arytenoidei interni or finally the adductor fibres may eventually become involved, and with the complete paralysis of the cords, which then assume the cadaveric position, respiration becomes less impeded, while the voice becomes impaired.

The fact that the voice is in no way impaired unless the internal tensors or adductors are involved, accounts for the fact that bilateral abductor paralysis may exist without the slightest suspicion on the part of the patient or physician. This is well exemplified by a case at the Bristol Royal Infirmary, that of a newsvendor, who had pursued his occupation till sudden respiratory embarrassment necessitated immediate removal to the Infirmary, where, in a second attack just after admission, he died before tracheotomy could be performed. The abductor paralysis was due to a moderately enlarged thyroid gland, and the postici muscles were found completely atrophied, so that, although this patient had been the subject of double abductor paralysis for a considerable period, he had had no symptoms sufficient to attract his notice till within a day or two of his death.

The degree of dyspnoea resulting from the narrowed glottic chink depends not only on the relative completeness of the paralysis, but also and quite as much on the slowness or rapidity of the development of the paralysis. While incomplete bilateral abductor paralysis of rapid onset is generally attended with very

considerable dyspnœa, it is remarkable that in the more slowly progressive affections, such as locomotor ataxia, the most complete paralysis with a chink so narrow that it is difficult to understand how it can suffice for respiration, may be present without the patient complaining of dyspnœa so long as he remains inactive.

Treatment.—If the condition, either bilateral or unilateral, is due to affections amenable to remedies, *e.g.*, syphilis, neuritis from diphtheria, cold, etc., they should be treated accordingly, while faradisation of the laryngeal muscles is steadily persisted in. Submucous injections of strychnine are sometimes useful in neuritic palsies. I have found nitrite of amyl, inhaled, of great service in relieving the dyspnœic attacks in a case complicating locomotor ataxia.

When the paralysis is due to compression of the recurrent laryngeal nerves in the neck, as, for instance, by an enlarged thyroid gland, it may be possible to remove the source of pressure by surgical interference. Lodge records a case in which the symptoms had existed for seven years, removal of the isthmus being followed by complete recovery from the abductor paralysis and consequent dyspnœa. But, of course, a considerable period must elapse between the removal of the compression and the recovery of motor nerve fibres; in Lodge's case the period was ten months.

But, as severe and fatal asphyxia may arise suddenly at any moment if the paralysis is complete and bilateral, the patient should either wear an intubation tube or tracheotomy should be performed. Intubation is only suited to cases in which recovery from the paralysis is possible; in all others tracheotomy is the only satisfactory procedure.

PARALYSIS OF THE THYRO-ARYTENOIDEI INTERNI AND EXTERNI.

THE INTERNAL TENSORS OF THE VOCAL CORDS.

Etiology.—This form of laryngeal paralysis, when unaccompanied by paralysis of the abductors, usually results from overstraining the voice, or may be due to catarrhal laryngitis, especially in anæmic and neurotic persons. Paralysis of the internal muscle often co-exists with or follows abductor paralysis, and may be due to any of the numerous causes of abductor paralysis, central or peripheral.

Symptoms.—The vocal cords cannot approximate perfectly, an elliptical space, extending throughout their length, being left on phonation, which consequently is weak, husky, or even lost. The vocal cords are practically the tendons of the thyro-arytenoidei interni muscles, which lie beneath the cords, and are inserted into the anterior two-thirds, their function being to render the free margin of the vocal cords tense and straight during phonation, coughing, etc., so that when they are paralysed the vocal cords lose their normal flat appearance, becoming rounded and narrowed.



FIG. 125.

Paralysis of the thyro-arytenoidei interni and externi during vocalisation.

It is most exceptional yet possible in a central lesion to have paralysis of the thyro-arytenoidei interni alone (of the muscles of the vocal cords), as in *Fig.* 126.

Treatment consists in rest, the administration of general nervine tonics, and the employment of the faradic current locally. This has often to be persisted in for a considerable time, and rarely fails to cure.

Any co-existent catarrhal condition must be treated with appropriate remedies.

The term *laryngoplegia* is sometimes applied to paralysis of all the muscles supplied by the recurrent laryngeal nerve. Complete paralysis may succeed paralysis of the abductors from any cause, provided the lesion (whether central or peripheral) implicates all the motor nerve fibres of the larynx, while such a lesion as division of the nerve of course results at once in complete paralysis.

In *laryngo-hemiplegia*, or total paralysis of one vocal cord (see *Fig.* 127), the respiration is hardly affected during rest. The voice is generally lowered in pitch, hoarse, and is readily tired. Sometimes the voice is lost, but ordinary conversation is generally possible, and sometimes the voice is almost normal when the healthy cord compensates by over-adduction. In male patients, at any rate, paralysis of one cord is often never



FIG. 126.

Paralysis of the thyro-arytenoidei interni in bulbar paralysis. Abduction was good in this case.

suspected, because it is erroneously assumed that *aphonia* must result from paralysis of a vocal cord.



FIG. 127.

Paralysis of the left vocal cord during phonation.

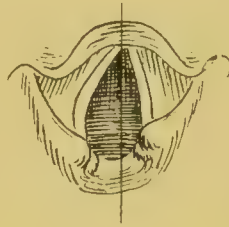


FIG. 128.

The same on deep inspiration.

During quiet respiration the larynx appears normal, but in vocalisation the healthy cord is over-adducted, and passes across the middle line to meet the paralysed cord, producing a peculiar distortion of the laryngeal image; the arytenoid cartilage on the paralysed side either remains immobile or, being unsupported by the muscles, it may be *pushed aside*, so as to lie behind the sound and over-adducted arytenoid. Like the paralysed vocal cord, the corresponding arytenoid appears to lie at a somewhat lower level than on the sound side, while its cartilage of Santorini is not tilted out on phonation.

On deep inspiration, as shown in *Fig. 128*, the arytenoid on the healthy side passes further *back*, as well as being more abducted than on the paralysed side. After the paralysis has existed for a considerable period, the arytenoid cartilage tends to become more or less fixed in the cadaveric position. The voice may get stronger in consequence.

In *bilateral complete paralysis*, or laryngoplegia, both vocal cords remain fixed in the cadaveric position, both during phonation and respiration. There is consequently complete aphonia and inability to cough, but there is no dyspnoea.

PARALYSIS OF THE ARYTENOIDEUS.

The arytenoideus may be paralysed alone in catarrhal conditions, from cold, or in hysteria.

A triangular chink is left between the vocal processes on attempted phonation, and the voice is generally lost. Local faradisation should be employed. It is combined paralysis of

the thyro-arytenoid and arytenoid muscles that produces the peculiar appearance in *Fig. 131*.



FIG. 129.

Diagram to illustrate the action of the arytenoideus.



FIG. 130.

Paralysis of the arytenoideus during vocalisation.



FIG. 131.

Combined paralysis of the arytenoideus and thyro-arytenoidei interni muscles.

DIFFERENTIAL DIAGNOSIS OF LARYNGEAL PARALYSES.

To summarise the various diseases which may cause laryngeal paralysis, they are :—

(1.) *Cortical lesions*.—Hysteria, organic lesions involving *on both sides*, the centres, or the motor fibres in their path from the centres to the medulla (necessarily very rare).

(2.) *Bulbar lesions*.—Nuclear degeneration (e.g., in syphilis, diphtheria, locomotor ataxia, general paralysis, disseminated sclerosis, amyotrophic lateral sclerosis, labio-glosso-laryngeal paralysis, syringomyelia, hæmorrhage and softening, tumours.

(3.) *Peripheral lesions*.—Pachymeningitis, intracranial new growths, new growths in the neck involving the vagus at the base of the skull, goitre, pericarditis, aneurysm of the aorta, right innominate, subclavian, or carotid arteries, intrathoracic tumours, cancer of the œsophagus, pleural thickening at the apex of the right lung, enlarged bronchial glands, injury to the nerves, neuritis, rheumatic, alcoholic, syphilitic and various tuberculous lesions, or those due to typhoid fever, lead, arsenic, phosphorus or other toxic causes.

(4.) *Inflammatory infiltration of the muscles*.

Paralysis of the larynx may be simulated by mechanical fixation of the crico-arytenoid joint.

Differential Diagnosis.—When the movements of one or both vocal cords are found to be impaired we should consider :—

Firstly, whether the motor defect is a true paralysis or is due

to ankylosis of the crico-arytenoid joint with more or less complete mechanical fixation of the arytenoid cartilage.*

Secondly, if it be a true paralysis or paresis ; whether the cause be (a,) local and myopathic ; (b,) due to a peripheral lesion of the motor nerve fibres to the larynx ; or, (c,) due to disease of the nerve centres.

Thirdly, what is the nature of the local, peripheral nerve, or central nervous disease, as the case may be.

Ankylosis of the crico-arytenoid joint is generally due to causes which result in infiltration of the surrounding tissues with obvious swelling or deformity. But sometimes the inflammation is of a more chronic character ; of the "adhesive" form with inflammatory degeneration rather than inflammatory exudation ; or again, ankylosis may result from simple long-continued inactivity in the joint following a neuropathic paralysis ; and it is in these two classes of cases that great difficulty may be found in coming to a definite diagnosis between ankylosis and true paralysis.

When the crico-arytenoid articulation is ankylosed on one side only, the laryngoscopic appearance very closely resembles laryngo-hemiplegia, but in unilateral recurrent paralysis, as we have already seen, the arytenoid cartilage on the paralysed side is displaced by the sound and over-adducted arytenoid cartilage, whereas in ankylosis this is not the case.

Attention to this point sometimes enables one to distinguish between a paralysed vocal cord and simple fixation of the cord from disease implicating the crico-arytenoid joint, for in the latter case the healthy arytenoid approaches but *does not displace* that on the affected side. This fact led me to diagnose simple arytenoid fixation in a patient that was said to have a thoracic aneurysm with resulting abductor paralysis, and whose laryngeal condition is shown in *Fig. 81*. The absence of aneurysm, after the laryngeal symptoms had existed for two years, was confirmed by two other physicians. Yet, in course of time, a simply paralysed cord becomes more or less fixed from disuse.

This patient had noticed huskiness of the voice two years before I saw him. His only other illness was diphtheria seventeen

* A vocal cord exhibits a peculiar heaviness of movement when the seat of infiltrating malignant growth ; this should not be mistaken for unilateral paralysis.

years previously, with paralytic sequelæ. He was reported perfectly strong and well five years later.

The Significance of a Laryngeal Paralysis is of considerable clinical importance, not only on account of the possible inconvenience from loss of voice, or danger of asphyxia from glottic stenosis (indeed, in most cases of partial paralysis of one cord neither vocal impairment nor any other laryngeal symptoms are present), but because the laryngeal condition may afford valuable aid in the diagnosis of various general diseases, and, in fact, for a long time may be the one and only definite physical sign pointing to the existence of grave organic diseases such as aortic aneurysm, locomotor ataxia, etc.

Isolated paralysis of the adductors, or the internal tensors of the cords, when bilateral, is generally due either to local catarrhal affections; or to functional cortical disturbances, such as hysteria; or else to general weakness or anæmia. Unilateral paralysis of the internal tensors, or paralysis of the inter-arytenoideus, is fairly certain to be of local origin, either catarrhal, tuberculous, or syphilitic.

Abductor paralysis may be due to organic disease of the vago-accessory nuclei or to pressure on, or neuritis of, any part of the recurrent laryngeal nerves, or of their nerve fibres in the vagi or within the medulla. It is therefore necessary to note any further symptoms indicative of bulbar paralysis, locomotor ataxia, syringomyelia, amyotrophic lateral sclerosis, or of a tumour involving the medulla or of syphilitic basal meningitis; and having excluded these affections, to consider whether the previous history of the case suggests diphtheria or syphilis as a possible cause of nuclear degeneration. Points in favour of a central nerve origin of the paralysis are: (1,) The paralysis being bilateral; (2,) Persistent frequency of the pulse (from implication of the inhibitory cardiac centre) without there being any pulmonary affection or rise of temperature to account for the increased pulse frequency; (3,) Co-existence of paralysis of the ocular, palatal, lingual or pharyngeal constrictor muscles. Having excluded central lesions, the examination should extend to the chest for symptoms pointing to aneurysm of the aorta or right innominate artery, pericarditis, cardiac hypertrophy, or to any growth in the anterior mediastinum, tuberculous deposits in the apex of the right lung, etc. With negative

results in these regions, the possibility of malignant disease of the œsophagus involving one or both recurrent nerves should be remembered. Naturally any obvious growth in the neck would be noted from the outset, but sometimes one is compelled, in the absence of any other discoverable cause, to attribute laryngeal paralysis to pressure on the recurrent by some apparently insignificant goitre or enlarged gland, which is only revealed by careful examination of the neck.

Again, the numerous causes of peripheral neuritis should be taken into consideration as possible sources of the paralysis.

The association of glottic spasm with laryngeal paralysis is usually due either to locomotor ataxia or to thoracic aneurysm, though the possibility of laryngeal crises in a case of laryngeal paralysis being a post-diphtheritic condition should be remembered.

A laryngeal crisis commences with a series of abrupt coughs resembling whooping cough, very different in character from the characteristic loud ringing brassy cough of aneurysm, and is followed by a long-drawn inspiratory whoop. A double abductor paralysis is more suggestive of locomotor ataxia than aneurysm of the aorta, but though a left-sided unilateral paralysis is very much more common in aneurysm of the aorta, bilateral paralysis has resulted in quite a number of cases.

The Laryngeal Manifestations of Intra-thoracic Aneurysm are of great clinical value and require careful consideration. The chief laryngeal symptoms are: (1,) cough; (2,) paroxysmal dyspnœa; and, (3,) alterations in the voice. These symptoms are the result of pressure on the pneumogastric nerves or their recurrent laryngeal branches.

On turning to *Plate XXXII*, the left vagus nerve is seen passing down in front of the arch of the aorta, and here its recurrent branch passes back beneath the aortic arch to ascend between the œsophagus and trachea. Obviously an aneurysm of the arch may cause pressure on either the left vagus or the left recurrent nerve, or on both, according to the situation and size of the aneurysm. Again on the right side, the vagus nerve may be seen descending in front of the right subclavian artery, and the recurrent nerve hooking round the artery to ascend behind the innominate artery so as to reach the sulcus between the œsophagus and trachea here. Plainly an aneurysm of the innominate

or subclavian arteries may cause pressure on either the right vagus or recurrent laryngeal nerve. But an aneurysm of the aortic arch involving that portion which gives off the innominate artery may reach sufficiently high up to cause compression of both the right and left vagus nerves, although this is obviously a less likely occurrence than is involvement of the left recurrent in aortic aneurysm, corresponding with the clinical fact that paralysis of the left recurrent laryngeal is far and away more frequent in aortic aneurysm than paralysis of the right nerve or of both recurrences. These remarks apply likewise to other intrathoracic tumours involving these nerves.

Now, if an aneurysm presses on one recurrent nerve, a purely motor nerve to the larynx, either unilateral spasm, or unilateral paralysis of the muscles of the corresponding side of the larynx results, according to the degree of pressure and its persistence. If *spasm*, probably all the muscles of that side are involved, but though dyspnoea is a prominent symptom, it is transient and not extreme, because the free movements of the unaffected vocal cord prevent total closure of the glottis during inspiration. If *paralysis* results, the abductors, in accordance with Semon's law, are first involved, and here neither quiet respiration nor the voice is altered. But with continued and increasing compression of the nerve, the internal tensor, and, sooner or later, the adductors of the cord are paralysed too. The result of this unilateral paralysis is impairment of the voice, for though the vocal cord of the opposite side can usually be over-adducted so as to compensate for the lack of movement in the paralysed cord, the increased exertion in talking resulting from the phonatory waste of breath and from the defective vibration of the flaccid cord speedily exhausts the patient; the voice, moreover, is usually wanting in tone, altered in pitch, impure and readily breaking into a falsetto. Catarrhal bronchitis and laryngitis are very frequently developed from pressure on a bronchus, and the voice is then more markedly impaired. Quiet respiration, however, is not interfered with, and there is no marked laryngeal stridor. A typical example of the order of events is afforded by a patient who came to my out-patient department at the Bristol Royal Infirmary, complaining of hoarseness. True it was that further examination elicited the fact that he had a cough and suffered from dyspnoea, but it was

the hoarseness that had attracted his attention. Examination of the larynx revealed abductor paralysis of the left vocal cord and the internal thyro-arytenoid muscle was weakened (*Fig. 132*), and this, in the absence of any appearance of local disease in a patient past middle life, pointed strongly to aneurysm. A few days later the internal tensor was like the abductor of the left side, completely paralysed (*Fig. 133*), and shortly after the adduc-



FIG. 132.



FIG. 133.

tors were paralysed too, resulting in complete paralysis of the left vocal cord. The *post mortem* examination subsequently proved the diagnosis of aneurysm of the aortic arch to be correct.

It is worthy of remembrance that paralysis may be absent at the first examination and yet rapidly develop within a few days from aneurysmal pressure.

But if the vagus nerve itself is compressed the course of events will be very different—the vagus, unlike the recurrent branch, containing centripetal as well as motor fibres. Slight compression may therefore cause spasm of both vocal cords—on the corresponding side by direct pressure, and on the other side, or rather on both sides, by the reflex irritation set up in the bulbar motor centres. Further, with increased or continued compression of the vagus its motor fibres may be paralysed in the invariable order already detailed, while spasm of the opposite cord may still be produced. Consequently, spasm of one cord and paralysis (abductor, or complete) of the opposite cord may co-exist. The result of vagus irritation is to cause paroxysmal attacks of coughing followed by urgent, but comparatively transient, dyspnoea and laryngeal stridor. If unilateral abductor paralysis has occurred, the symptoms are precisely the same, but if the opposite recurrent is also involved,

and double abductor paralysis is associated with laryngeal spasm, the resulting dyspnœa compels the patient to make violent inspiratory efforts, and, even after the spasm has relaxed, the vocal cords are sucked together and prolonged or fatal asphyxia is a probable consequence.

With bilateral complete paralysis the voice is lost, but dyspnoea can no longer be a consequence of laryngeal obstruction, though it may then, and at all times in cases of aneurysm or of tumours, result from pressure stenosis of the trachea or main bronchi.

The cough in aneurysm of the aorta has certain peculiarities so distinctive that the existence of aneurysm may often be suspected from the character of the cough alone. In the earlier stages of vagus pressure and irritation, the paroxysms of coughing are associated with glottic spasm and the cough is loud, ringing and brassy. When complete unilateral or bilateral recurrent paralysis supervenes, the ventricular bands are imperfectly apposed on coughing, consequently the cough loses its explosive character and becomes wheezy, or, as Wyllie aptly describes it, "bovine"—like the cough of a cow, which has no false cords or ventricles of Morgagni.

Tracheal tugging, described by Porter, is a valuable sign in certain deep-seated aneurysms due to the pulsations of the aneurysm being communicated to the left bronchus and trachea by the transverse portion of the arch. In examining for the presence or absence of this sign, the patient should be made to sit up with the head inclined forward so as to relax the neck completely. The trachea should then be put upon the stretch either by firmly grasping the cricoid cartilage between the forefinger and thumb and exerting upward pressure; or in males it is sufficient to exert upward extension by the finger placed below the projection of the thyroid cartilage. This sign is never present in health, and though very rarely it may be due to a mediastinal growth lying between the arch and the bronchus, or pressing the arch against the bronchus, its existence points very strongly to aneurysm of the posterior portion of the aortic arch.

MUTISM.

Inability to *articulate* sounds is termed mutism or dumbness, and is due to functional inactivity of the speech centre in Broca's

convolution. Such functional inactivity may be the result: (*a*), of idiocy or dementia, or of destructive lesions in the centre or involving the motor nerve fibres in their course downwards as far as the bulbar centres, *e.g.*, in the corona radiata, internal capsule, etc.; (*b*), of functional neuroses, *e.g.*, hysteria, etc.; or, (*c*), it may be due to deafness. In none of these conditions is there laryngeal paralysis in the true sense of the word, for reflex movements of the vocal cords, *e.g.*, in coughing, are unimpaired, but *voluntary* movements of the vocal cords in speech only are in abeyance.

Gross cortical lesions belong entirely to the realm of neurology, and need not concern us here.

Hysterical mutism may be regarded as a more aggravated or complete form of hysterical aphonia, the inability to phonate being extended to the inability to whisper. Like hysterical aphonia the onset is generally sudden, and is often associated with a fright or some severe emotional disturbance. It is sometimes associated with various hysterical stigmata.

The oral mechanism, as well as the laryngeal, is necessary for whispering, and in hysterical mutism there is paralysis of the whole speech mechanism, while in hysterical aphonia, the laryngeal mechanism alone is involved. In a case of hysterical mutism reported by Semon there was paralysis of the adductors of the vocal cords, and one energetic intralaryngeal faradisation not merely cured the functional paralysis, but also restored to the patient the power of articulate speech.

Apsithyria is a term applied by Solis Cohen to an inability to whisper, although ordinary speech is performed normally.

DEAF-MUTISM.

Etiology.—Deafness in considerable degree, congenital or acquired in the very young, interferes with the acquisition or development of speech and results in “deaf-dumbness” or “deaf-mutism.” In the ordinary deaf-mute the vocal cords and the whole motor speech mechanism is anatomically normal and well developed, but inability to hear words renders the speech centres functionally inactive. The deafness varies in degree, but what degree of deafness will result in deaf-mutism cannot be defined. Love and Addison define the word “surdism” as that degree of deafness which makes the acquisition of

speech in the very young impossible by ordinary means, or which involves the loss of recently-acquired speech. In different countries the ratio of deaf-mutes to the general population varies. Thus, in Prussia, France, and in Great Britain and Ireland it varies from about 1 in 1,730 to 1 in 1,630, and in North America it is about 1 in 1,250. The lowest average is found in Holland (about 1 in 3,000), and the highest in Switzerland (about 1 in 900).

It has been found that the average deaf-mute up to the age of thirteen, is in all respects except the deafness, equally developed physically in the earlier years of life, as the normal boy or girl, provided the deaf-mute be placed under similar hygienic conditions and properly fed. But Love and Addison found the deaf mute boys at fifteen years of age were inferior in most respects to the hearing boys of the same rank of life, the "head measurements being nearly half-an-inch less than in the hearing boys." A certain proportion of "deaf-mutes" are aphasic or idiotic, but in these the mental defects are due to the defects of the defective central nervous system generally, and not to the deafness *per se*, and these feeble-minded children are, of course, not amenable to the special training which is called for in the child who is deaf and therefore dumb, but otherwise physically well developed.

Pathology.—The deafness may be congenital, and congenital deaf-dumbness is prone to occur in the children of congenitally deaf-dumb parents, or in the children of parents whose family history shows a tendency to deaf-mutism. Marriage by such individuals is therefore undesirable. But in a large proportion the deafness ("surdism") is acquired, and may be due to any of the numerous causes of acquired deafness, especially cerebral inflammation, meningitis, convulsions, and the exanthemata. Love and Addison state that "if the disease which destroys the hearing happens before six or seven years, mutism will be apt to follow. The onset of the latter will depend: (*a*), on the amount of speech previously learnt; (*b*), on the damage caused to general intelligence by the disease producing the deafness; (*c*), on the efforts used for the preservation of speech already acquired, and for the extension of the deaf child's vocabulary."

In one hundred consecutive entries at the Bristol Institute, seventy-one were described as "congenital" and twenty-eight as "acquired" (one not classed).

The defects causing deafness in the congenital cases are in a small proportion due to malformation in the external and middle ear. In the larger number they are the result of arrested development of the structures forming the internal ear, generally due to inflammatory new formations by extension of disease (*a*), from the middle ear, or (*b*), from the central nervous system.

Acquired deaf-mutism is generally due to inflammatory disease of the internal ear secondary to otitis media, with involvement of the membranous labyrinth and its nervous structures. Evidence of existing or former disease of the middle ear is to be found in a large percentage of deaf-mutes; and in a considerable number post-nasal growths, collections of cerumen in the external meatus, and other removable causes of deafness are present.

Other cases are due to meningitis, and, rarely, to cortical lesions.

To summarise: Congenital cases are due to developmental errors. Acquired cases are due to: (1,) Central lesions from meningitis and cortical lesions; (2,) Panotitis during some fever, etc.; (3,) Marked impairment of hearing due to adenoids or other removable causes.

Deaf-mutes may be classified according to the degree of deafness, thus: (*a*), Totally deaf—congenital deaf-mutes; (*b*), Semi-deaf—those whose deafness is acquired before speech; and, (*c*), Semi-mute—those whose deafness is acquired soon after speech has been acquired. The degree of deafness should be tested: (1,) by the loud voice close to the ear, and by a loud bell for aërial deafness; and, (2,) by a large tuning fork for bone conduction.

In forty-five cases at the Bristol Institute, examined by Mr. Harsant and myself, we found that twenty-seven, or 60 per cent., were totally deaf.

Very interesting statistics bearing on hereditary deafness have been collected by Fay from inquiry into over 4,000 marriages of deaf persons in the United States (abstract, "The Messenger," Jan. and Feb., 1900):—

In 3078 marriages of deaf persons, 9·7 per cent. resulted in deaf offspring. Of 6782 children born, 8·67 per cent. were deaf.

In 2377 marriages, both partners deaf, 9·25 per cent. resulted in deaf offspring. Of 5072 children born, 8·46 per cent. were deaf.

In 599 marriages, one partner deaf, 12·52 per cent. resulted in deaf offspring. Of 1532 children born, 9·86 per cent. were deaf.

In 335 marriages, both partners congenitally deaf, 24·78 per cent. resulted in deaf offspring. 25·93 per cent. of the children were deaf.

In 814 marriages, one partner congenitally deaf, the other adventitiously deaf, 8·11 per cent. resulted in deaf offspring. 6·54 per cent. of the children were deaf.

In 120 marriages, one partner congenitally deaf, one partner hearing, 14·66 per cent. resulted in deaf offspring. 11·93 per cent. of the children were deaf.

In 845 marriages, both partners adventitiously deaf, 3·55 per cent. resulted in deaf offspring. 2·33 per cent. of the children were deaf.

In 310 marriages, one partner adventitiously deaf, one hearing, 3·23 per cent. resulted in deaf offspring. 2·24 per cent. of the children were deaf.

In 1775 congenitally deaf partners, 82·36 per cent. had deaf relatives (not descendants).

In the adventitiously deaf partners, 31·75 per cent. had deaf relatives.

Of the hearing persons who married deaf partners, 45 per cent. had deaf relatives.

Of marriages where both were congenitally deaf, and both had deaf relatives, 28·49 per cent. resulted in deaf offspring, and 30·10 per cent. of the children were deaf; when one had and one had not deaf relatives, 16·33 per cent. resulted in deaf offspring, and 20 per cent. of the children were deaf; but where neither partner had deaf relatives, the percentages were only 7·14 and 4·17.

Of marriages where both were adventitiously deaf, and both had deaf relatives, 17·54 per cent. resulted in deaf offspring, and 9·65 per cent. of the children were deaf.

Such figures as I have quoted will form some guidance to those called upon to give advice as to marriage amongst those who are deaf, or have a family tendency to deafness.

Treatment.—When a deaf-mute child is brought for our opinion, the general condition of mental and physical development should first be considered. Inability to speak or to understand what is said to it, may be due to imbecility apart from deafness, though the child may or may not be deaf. As already stated, these cases are not, properly speaking, cases of deaf-mutism. On the other hand, it is necessary sometimes to exercise caution lest a well-developed child who, owing to early deafness, has had little chance of developing mentally, and whose training has been much neglected, be regarded as idiotic.

It is important to recognise cases of incipient progressive deafness, where there is sufficient hearing power still left to make the acquisition of speech possible ere the deafness becomes too pronounced, otherwise invaluable time may be lost from our failing to impress the parents with the true facts of the case, and leaving them with a delusive hope that the child may grow out of the deafness as it gets older.

Again, it is our duty to examine the patient with a view to differentiating those cases in which the deafness is purely central, and those in whom internal deafness is complicated and increased by removable causes. For instance, post-nasal growths are present in a large number of deaf-mutes, and though these adenoid growths are very rarely the chief cause of the deaf-mutism, inasmuch as they rarely develop sufficiently early in life as to cause deafness of such a degree as to prevent the acquisition of speech, yet they may so far increase central ear deafness as to cause the child to become a deaf-mute. Lake records a striking instance of cure of deaf-mutism by the removal of adenoids. More frequently have such removable factors in the deafness a practical importance on account of their interfering with the application of the oral method in subsequent training. It is desirable, therefore, that removable causes of deafness should receive appropriate treatment, while otitis media purulenta and other diseased states that may be present naturally demand treatment on account of their menace to life or general health.

Finally, we may be able to suggest the best method for the training of the deaf-mute. There are many methods or systems taught at present, the chief being known as (*a*), the pure oral or German method; (*b*), the sign and manual or French method; (*c*), the manual; (*d*), the combined oral and sign-manual; and, (*e*), the auricular method. The relative advantages of the different systems are very difficult to estimate, as the advocates of each different method are so often hopelessly prejudiced in favour of their own particular system to the exclusion of others. The fact is that no one method is the best for all cases. Our main object is to teach language, that is, the means by which thoughts and ideas are communicated. Amongst savages language consists largely of signs and gestures; in civilised communities these are almost wholly replaced by spoken words or written words, and the deaf-mute being naturally deprived of acquiring the meaning of spoken or written words, resorts to the more primitive methods of sign language. The sign method can be utilised for the acquirement of the manual and written language. Nevertheless, if the oral method of speech and lip-reading can be acquired by a deaf-mute, together with that of writing, he is obviously placed in a much better position for

intellectual intercourse and social usefulness than he can be by any of the other methods. Unfortunately it takes a long period of teaching before the oral method can be utilised for mental and intellectual development, and in a considerable proportion is so imperfectly acquired in the end that it is of no practical use to the individual. Now it is very difficult to determine whether a child is capable of profiting by the oral method until it has been tried, natural aptitude depending partly on the intellectual capacity, and partly on the powers of observation, a very variable quantity even in normal children of equal intelligence. On this account, the late Royal Commission favoured the dual system in schools, that is to say, schools where both the oral method and the sign and manual methods are taught separately, so that a child which is found unsuited to the oral method may be turned over to the other teachers.

Speaking generally, the congenitally total deaf cases do not lend themselves to the oral method, and their voice is often harsh, disagreeable, and unintelligible, as there is no perception of sound to assist them in modulating its tones, nevertheless many of them are quick and intelligent, and yield very successful results. Those, on the other hand, who have some hearing power, acquired semi-deafness, start with a distinct advantage for oral teaching, yet in not a few the intellectual powers have been impaired owing to concomitant damage to the brain by the disease which has caused the deafness.

For the semi-deaf, the auricular method may be applicable. I refer to the employment of the modified telephone invented by Dr. Thornton and utilised with some success by Barrett in the Margate school.

I have endeavoured to show how much careful consideration is needed in giving sound advice in dealing with a deaf-mute child, and to caution against lightly-uttered opinions where grave issues are at stake.

Without presuming to dogmatise on what must at present be an open question, I am of opinion that :—

(a.) For the working-class deaf-mute who will have to earn his living by mechanical labour, but who cannot hear the loudest voice close to his ear, the sign-manual system is generally most suitable. He has only a certain number of years for mental training, and so long a time is taken up in acquiring

the oral method that he has insufficient opportunity for acquiring facts and ideas.

(*b*.) For the better-class child who has some slight hearing power, and can be kept under special training for at least eight years, the oral method should be adopted first, and the combined oral and sign manual system superadded.

(*c*.) For deaf-mutes who can hear consonants and a few vowel sounds of the loud voice, or who can hear the sound of their own voice, the oral method, or the combined method is most desirable.

A child should be brought under special training not later than the seventh year. There are many advantages in well-managed institutions over day-classes, for instead of having its special training during school hours only, the child lives all day in an environment of perpetual training.

THE THROAT AFFECTIONS OF CHRONIC DISEASES OF THE NERVOUS SYSTEM.

Locomotor Ataxia.—The pharyngeal mucous membrane may be the seat of hyperæsthesia, anæsthesia, or paræsthesia. Various paralytic phenomena have likewise been observed, *e.g.*, right-sided paralysis of the soft palate (Dundas Grant).

It is to the labours of Semon and Burger that we are chiefly indebted for our knowledge of the laryngeal affections in *locomotor ataxia*, in which four kinds of laryngeal affection may occur: (1,) *Sensory disturbances*, anæsthesia, hyperæsthesia, paræsthesia. Various abnormal sensations in the larynx precede or accompany laryngeal crises, and in a few cases persistent anæsthesia has been reported. (2,) *Inco-ordination* of the laryngeal muscles, or laryngeal ataxy. The voice may be jerky, or resemble dysphonia spastica in being interrupted by intervals of aphonia, or huskiness. Fournier states that in ataxy of the cords the patient suddenly and unexpectedly loses his voice, or at least the power of distinct articulation, and the voice becomes thick, dull and discordant. Krause describes the vocal cords as being suddenly approximated, then remaining still in a semi-adducted position, and then approximated in the median position. During abduction the cords, after being driven together with great force, recoil into the most extreme inspiratory position. Burger observed irregular movements of the vocal

cords during regular and deep respirations, that is, the vocal cords executed two or three incompleting movements of abduction and adduction. It is a rare condition, and only one case have I seen in which the abductor and adductor movements of the cords were ataxic, and even here the symptoms were not marked. (3.) *Laryngeal crises* are frequently present in locomotor ataxia. According to Gowers they are almost as frequent as gastric crises. They may constitute the earliest manifestation of locomotor ataxia, and in a large proportion of cases are associated with abductor paresis, but usually laryngeal crises do not occur till the ataxic symptoms are well established. Semon attributes the onset of the crises to an increased latent irritability of the adductor centres, excited by some peripheral irritation, as for instance drinking a glass of water, touching the larynx with a probe, etc. The patient feels a sense of tickling or dryness, or of stricture in the laryngeal region, quickly followed by a succession of abrupt coughs—resembling whooping cough. These continue till the patient feels almost asphyxiated, and are followed by inability to inspire, or by a long-drawn whoop, during which air is drawn into the chest with very great difficulty. The whole attack may last but a quarter of a minute, or may persist for five or ten minutes. Death from asphyxia is unusual, but has occurred. In some cases the laryngeal crisis is attended by loss of consciousness, vomiting, vertigo, or pains in the chest or limbs. (4.) *Paralysis*, usually of the abductors, of the cords. Burger collected seventy-one cases recorded up to the year 1891, and in forty-three of these there was either unilateral or bilateral paralysis of the abductors. The symptoms of abductor paralysis are described on p. 257. After abductor paresis has lasted for some time, it may be followed by the supervention of adductor paralysis. But abductor paralysis of the vocal cords may be the first and for a long time the only indication of commencing locomotor ataxia. Semon has recorded an instance of abductor paralysis existing for twelve years prior to the development of general symptoms. Dundas Grant showed a case at the Laryngological Society of London, which when seen two years before had almost complete abductor paralysis, with right-sided paralysis of the palate. At the time he was shown his epiglottis was pendulous, and he had paralysis of the abductors and both

internal and external tensors of the cords, but no paralysis of the laterales.

In some cases of tabes dorsalis with abductor paralysis the pulse rate remains abnormally frequent. It is probably due to the fact that the inhibitory nerve of the heart is, like the motor nerves of the larynx, derived from the vago-accessory nucleus, and thus *paresis of the abductor muscles, associated with persistent increased pulse frequency*, should always lead to the suspicion of tabes.

General Paralysis.—Kellogg states that in ascending general paresis of the insane tonic œsophageal and pharyngeal spasm is not very rare; laryngeal spasms also occur. He has recorded a case of laryngeal spasm continuous for two years, as a premonitory symptom of general paresis which finally terminated fatally in a typical form. Clonic lingual spasm, too, is not a very rare symptom.

At the Bristol meeting of the British Medical Association, 1894, Dr. Permewan communicated the result of the laryngoscopic examination of thirty-four cases of general paralysis of the insane, and concluded that in at least 20 per cent. of these cases there was more or less marked posticus paresis. His observations confirmed the general truth of Semon's law as to the particular susceptibility of the abductors to succumb to organic disease. It is interesting to note that in Permewan's cases there was no regular combination of symptoms pointing to the existence of posterior sclerosis, although there is undoubtedly such a close relation between general paralysis and tabes dorsalis.

Labio-glosso-laryngeal Paralysis.—Loss of the pharyngeal and laryngeal reflex has been noted by Kussmaul. The absent reflex and consequent entry of food particles into the trachea was due to motor changes, not to anæsthesia, as there was no loss of sensibility in the pharynx or larynx. Laryngeal crises are almost unknown in this affection, but there are several recorded cases of abductor paresis. I have met with one instance (*Fig. 126*) in which apparently thyro-arytenoid paralysis was present alone, without any abductor paresis, although the other usual features in the tongue and soft palate were well marked. Permewan, in the paper referred to above, related the history of a case in which paresis of the adductors was observed with complete bilateral abductor paralysis within nine months

of the commencement of abductor paresis. De Havilland Hall, in one case, observed isolated paralysis of the interarytenoideus.

Disseminated Sclerosis.—Laryngeal paralysis is very rare in "*sclerose en plaques*," but the vocal cords in one case under my care exhibited fine irregular tremor during phonation. In another case the vocal cords showed interrupted movement on abduction or adduction, and clonic movements on receding to the position of quiet respiration. Collett has observed pronounced oscillations of the vocal cords during respiration, with tremor of the cords on phonation, and in one case Horne noted incomplete anæsthesia and paralysis of the left side of the soft palate, and abductor paralysis of the left cord. The slow, monotonous tone, with jerky voice and scanning speech, is an early feature in most cases.

The nasal mucosa may be hyperæsthetic; and slow, quiet destruction of the septum and hard palate (Barr, and trophic ulceration of the *alæ nasi* (Giraudeau), have been recorded.

Syringomyelia.—Both motor and sensory troubles of the pharynx and larynx occur, but the sensory disturbances are most frequently seen. Dysphagia may be complained of, due to more or less pronounced paresis of the muscles of the soft palate. Paralysis of the right side of the palate and pharynx, associated with paralysis of the right cord has been observed (Tilley). Eighteen cases in the practice of various French rhinologists have been investigated by Cartaz, who found that motor or sensory troubles in the larynx occurred in about half the cases. Paresis or paralysis of the abductors was found three times, and paralysis of the recurrent nerve twice. Thermic sense was modified independently of ordinary sensation in some cases, and paralysis, anæsthesia, or thermic sense abolition was sometimes bilateral, in other cases unilateral. The onset of all the lesions was insidious. The sensory troubles often passed un-noticed, and were detected only on systematic examination. Patients with syringomyelia may show laryngeal symptoms as early as the seventh year.

No cases of laryngeal crises in syringomyelia appear to have been observed.

The nasal mucosa was anæsthetic, without disturbance of the olfactory sense in three of Cartaz's collected cases.

Paralysis agitans.—The speech of a person with paralysis agitans has been compared by Charcot to that of a bad horseman trying to talk while riding a high trotting horse. Müller states that the voice is tremulous and interrupted by intervals of silence. He observed that abduction of the cords after phonation was interrupted by two or three imperfect adductor movements, and Rosenberg found that in phonation the cords failed to be maintained in the median position more than a brief period.

Acromegaly.—The following conditions were noted by Chappell in a case of acromegaly. The anterior and posterior pillars, the soft palate and the uvula were much thickened, also the tonsils and their capsules. The lingual glands were much hypertrophied. An external examination showed that the larynx was very much enlarged. The epiglottis was thickened, and the arytenoid cartilages and the ventricular bands were enlarged. The opening between the vocal cords was very small. While the patient remained quiet, respiration was very slightly impaired, but excitement produced laborious breathing and a crowing sound during both expiration and inspiration, and during one of these attacks of dyspnœa the patient died.

The inferior turbinated bodies were enormously enlarged ; the other structures in the nasal cavity appeared normal.

CHAPTER XII.

RHINITIS.

ACUTE, PURULENT, CHRONIC, HYPERTROPHIC, ATROPHIC AND FIBRINOUS
RHINITIS—RHINITIS CASEOSA—RHINOLITHS—XANTHOSIS.

ACUTE CATARRHAL RHINITIS.

Simple Acute Rhinitis, the common cold in the head, hardly requires lengthy notice here. There is good reason to believe that it is due to a micro-organism affecting a susceptible mucous membrane, having an incubation period of about two days, and which, in individuals predisposed, acquires increased virulence; thus it is we often see one weakly member of a family who is continually catching colds in the head and infecting the other less susceptible members in turn.

It is unnecessary to describe the well-known symptoms of simple nasal catarrh, but it is always well to remember that acute rhinitis is a common symptom in the early stages of measles and influenza, and that the symptoms of vaso-motor rhinitis, hay-fever, nasal polypus, etc., may very closely simulate a simple cold in the head. The muco-purulent discharge of the later stages of simple catarrh may, without due care, be mistaken for a true purulent rhinitis.

Complications.—Even in simple nasal catarrh, and more frequently in influenzal, scarlatinal, and morbilliform rhinitis, the inflammation may spread to the nasal accessory sinuses, causing severe pain in the forehead, cheek, or in the nose, together with other indications of acute sinusitis, which is fully described in the section on “Accessory Sinus Disease.”

Treatment.—Practical experience has shown that it is possible to abort a cold if it be taken in time, and most of the remedies that have proved successful are local stimulants to the nasal and pharyngeal mucosa, or local germicides. For those who can take it there is no remedy which is so effectual in aborting an

incipient catarrh as quinine in doses of 15 or 20 grains during the first twenty-four hours. A hot water and mustard foot-bath, and the administration of 10 grains of Dover's powder together with hot drinks on going to bed, is a popular device which often proves successful. Lederman, of New York, has found the following mode of treatment very beneficial during the congestive stage of an acute nasal catarrh. The nasal chambers are sprayed with any of the antiseptic solutions, Seiler's preferred, until they are sufficiently cleansed, and then the following solution is used:—

℞ Cocaine						
Menthol āā	-	-	-	-	-	grs. xx
Benzoinol	-	-	-	-	-	fʒij
M. ft. solutio.						

Capitan has recommended the frequent insufflation of the following:—

℞ Pulv. talc	-	-	-	-	-	grs. lxxv
Antipyrin	-	-	-	-	-	grs. xv
Acid boric (pulv.)	-	-	-	-	-	grs. xxx
Acid salicylic	-	-	-	-	-	grs. iv

I have frequently prescribed Formula 49 with good effect.

Attention to the general health and general hygienic surroundings, exercise in the open air, and the daily use of the cold bath, or cold bathing, will do more than any local treatment towards rendering the patient more resistant and less liable to colds. Acute rhinitis may be simply the result of a chill, a vaso-motor rhinitis, the symptoms of which come on in a few hours after exposure, and are followed by a simple catarrh which may pass off in a day or two, or may often be checked effectually by a Turkish bath, or a hot bath and a Dover powder on going to bed.

PURULENT RHINITIS.

The acute purulent rhinitis of infants, *rhinitis neonatorum*, is generally due to infection at birth by leucorrhœal or gonorrhœal discharge in the maternal passages, and is therefore similar in source and character to purulent conjunctivitis. In other cases the nose becomes inoculated after birth.

In children especially, a form of chronic purulent rhinitis is sometimes associated with the strumous diathesis, the mucosa being swollen, boggy, and bathed in thick, viscid muco-pus.

The micro-organisms usually present are gonococci and the various pyococci. In one case a discharge of blue pus from the nose was due to the *b. pyocyaneus*.

The pathological changes in the mucous membrane are similar to those observed in suppurative inflammations of other mucous membranes, but the thickening of the submucosa, firstly from hyperæmia, and secondly from cell proliferation, causes more or less pronounced nasal stenosis. Moreover, there is always a possibility of involvement of one or more of the accessory cavities of the nose, giving rise to a train of fresh symptoms which are excluded from consideration here.

The persistence of the inflammatory affection may result in a chronic hypertrophic rhinitis, and it is possible that with subsequent resolution atrophic rhinitis may form a final stage in the course of events.

The **Diagnosis** involves two main questions: (*a*,) Does the pus really come from the nasal passages, or from the accessory cavities? (*b*,) If from the nasal passages, is it primary or secondary to a growth, foreign body, or some infective disease?

A purulent discharge from the nose may occur also in syphilis, tuberculosis, diphtheria, measles, small-pox, erysipelas, glanders, etc., of the nose. Foreign bodies, rhinoliths, new growths, etc., often set up purulent rhinitis, but the discharge is then usually unilateral; and a traumatic abscess of the septum may be either unilateral or bilateral.

Finally, the possibility of the discharge coming from one of the nasal accessory sinuses must always be borne in mind.

Another but rare cause of purulent nasal discharge in infants is acute osteomyelitis of the upper jaw (Schmiegelow). The eyelids become swollen and œdematous, and suggest gonorrhœal ophthalmia, but the swelling of the cheek, hard palate, and the falling out of dental buds, with alveolar pyorrhœa, should prevent mistakes in diagnosis.

The **Treatment** of purulent rhinitis in infants may become an urgent matter, owing to the impossibility of their taking the breast so long as nasal respiration is interfered with. The nasal passages should be syringed frequently with some mild warm antiseptic lotion, and subsequently a 1 or 2 per cent. solution of protargol in water and glycerin should be applied to the nasal passages by means of a small camel-hair brush.

In other cases the special treatment will largely depend on the actual cause of the purulent discharge, but in most it is necessary to use some alkaline and antiseptic spray or douche, to clear away the crusts and masses of secretion. For this purpose, Dobell's solution, or one of the formulæ given at the end of the book, may be employed.

CHRONIC RHINITIS.

Etiology.—Simple chronic rhinitis may result from frequently recurring attacks of acute rhinitis, which results in a certain degree of permanent thickening of the tissues and chronic congestion—a more or less persistent chronic rhinitis with frequent slight or acute exacerbations. Chronic rhinitis is thus often left in children after measles or scarlet fever, and is associated with more or less ill health, a condition which should not be neglected or treated too lightly, as it so often leads to the implication of the middle ear. Similarly the strumous diathesis or the existence of post-nasal adenoids are common causes of chronic rhinitis and of ear disease. Doubtless many cases of otitis media in children could be prevented by timely treatment of chronic rhinitis.

In adults we find other causes of chronic rhinitis in the inhalation of irritating particles of dust, such as are produced by working in stone, mattress making, and upholstery. There are, however, numerous less obvious causes of chronic rhinitis such as dyspepsia, constipation, portal congestion, excessive use of alcohol, sexual excess, anæmia, and the gouty diathesis; in other words, almost all the causes of chronic pharyngitis are possible causes of chronic rhinitis. French rightly urges that functional disorders of the digestive tract are capable of producing vaso-motor reflex irritation of the inferior turbinated bodies, as is evidenced during attacks of acute gastric or duodenal catarrh.

Rhinitis sicca is a chronic rhinitis attended with deficient secretion, the mucus becoming inspissated and tenacious, or forming simply dry crusts of mucus unattended with fœtor. It is found chiefly in anæmic girls, or as a manifestation of the gouty or rheumatic diathesis, or may be due to chronic alcoholism. It is often associated with pharyngitis and laryngitis sicca.

Symptoms.—The only marked symptom is constant nasal discharge; in elderly patients especially this may be simply a copious watery exudation. The nasal mucous membrane is sometimes congested and the turgid turbinal bodies pit on being touched with a probe. These cases should perhaps be regarded rather as a form of vaso-motor rhinitis. When the nasal discharge is due to irritation of particles of dust or, as in children, is a sequence of the exanthemata, the discharge is usually muco-purulent and strings of sticky mucus occupy the nasal passages.

Treatment.—*General.*—In dealing with the chronic forms of rhinitis, attention to the general health is, of course, essential to success. It is too often regarded as a purely local affection. Indigestion, a tendency to constipation or torpidity of liver, if present, should be treated. The possible existence of post-nasal adenoids should be borne in mind. Change of air or a voyage, out-door exercise, avoidance of hot, overcrowded rooms and late hours, will often succeed when local treatment alone has failed; and when we remember that chronic rhinitis is often largely dependent of disturbance of the neuro-vascular mechanism, that it is sometimes almost a neurosis, the necessity for a generous tonic line of treatment is obvious. Massage and cold douching daily are most beneficial, and I may cite the case of a medical friend of mine who, having suffered from chronic rhinitis for years, and in whose case local treatment had been attended with no lasting improvement, underwent a course of Turkish baths and massage continued for several months in succession, and has remained perfectly free from his nasal trouble for many years. Many similar instances have occurred in my experience.

Beverley Robinson recommends a tablet containing gr. $\frac{1}{4}$ each of chloride of ammonium and powdered cubeb with some liquorice, together with codeine if there is much cough, taken every fifteen or thirty minutes, or every hour for some time.

Local.—In the earlier stages of chronic rhinitis, such general measures may be aided by mild local applications. Supra-renal capsule extract in the form of a spray or a powder mixed with boracic acid, and gum acacia, used as a snuff, will act as an admirable local astringent. I have also found great benefit from liquid vaseline containing terebene (℥ x ad ̄j), eucalyptol (℥ xv-xx ad ̄j), with camphor (gr. i-ij ad ̄j) sprayed well into the

nose with an oil atomiser. This should be done night and morning for some weeks. If crusts of dried secretion tend to collect in the passages, a weak solution of bicarbonate of soda and borax in warm water should be forcibly sprayed in for some minutes till they are loosened and can be cleared out, the oily solution being subsequently used. A post-nasal spray must be used occasionally by the medical attendant, to ensure thorough removal of all collections of inspissated secretions in the nasopharynx.

As the condition improves, and the mucous membrane of the nose becomes more tolerant, a snuff composed of sodium chloride ʒij , boracic acid ʒss , ammonium chloride ʒss , camphor gr. j , may be used twice daily in the place of the other local remedies.

These are the remedies I have found most generally useful, but many others are recommended by various authorities, such as solutions of zinc sulphate grs. ij , alum grs. iv to $viii$, zinc chloride gr. j , nitrate of silver, grs. ij to xv , or sodium benzoate grs. xxx to the ounce; tar water; insufflations containing nitrate of silver, tannic acid, iodol, soziodol, potassium soziodolate sanguinaria, etc.



FIG. 134.

Method of using the Auto-insufflator.

CHRONIC HYPERTROPHIC RHINITIS.

Etiology.—This may be regarded as an advanced stage of simple chronic rhinitis, and may, therefore, be due to those conditions which lead to chronic rhinitis. From the histological investigation of twenty cases Wyatt Wingrave found almost invariably mucoid degeneration of the muscular walls of the venous sinuses, and he suggests that in the persistent distension of the sinuses which ensues we have the explanation of the general hyperplasia associated with “turbinal varix.”

Symptoms.—The nasal obstruction resulting from the hypertrophy of the tissues, and the constant presence of thick

tenacious mucus which passes into the naso-pharynx and leads to constant hawking, are very distressing to the patient. The symptoms, in fact, are those of simple chronic rhinitis greatly intensified, the obstruction of the nasal passages being often so considerable that the patient cannot blow his nose properly.

Particularly in this form of rhinitis are reflex nasal neuroses prone to occur, especially a persistent, hard, spasmodic cough, analogous to that cough which is excited by the passage of the Eustachian catheter. The existence of hypertrophic rhinitis will sometimes increase a liability to asthma and hay-fever.

On *examination* of the nasal passages, we find a general thickening of the mucous membrane. Especially is this noticeable in regard to the lower turbinals; these become greatly thickened from the overgrowth of fibrous trabeculae between the venous spaces, which are more or less obliterated. The nasal passages are often greatly narrowed by these hypertrophied inferior and middle turbinated bodies.

With the rhinoscopic mirror the inferior turbinals are seen to have become even more hypertrophied posteriorly, the pale greyish-white, mulberry-like extremities more or less blocking the choanae (see *Plate XVIII, Fig. 3*). Muco-purulent secretion is usually present, and if abundant may even have to be removed by a coarse alkaline spray, in order to see clearly the condition of the nasal structures. These hypertrophied turbinals do not collapse to the normal extent on applying cocaine or suprarenal extract, and thus we have a ready means of distinguishing hyperplastic overgrowth from simple venous distension of these normally vascular structures.

There should be no difficulty in distinguishing the pale-grey fibrous-looking hypertrophied turbinal bodies from a mucous polypus, which is moveable, soft, and semi-translucent. In addition to the abnormal conditions already mentioned, we very often find a narrowing of the nasal passages from a deviation of the septum, or a spur, or some other structural deformity which has had some share in setting up the conditions which lead to hyperplastic changes in the nasal mucosa.

Treatment.—Our aim should be to reduce the volume of the enlarged turbinates, and when we have to deal with a well-marked hypertrophic form of rhinitis, it is necessary to have recourse to operative measures, for it is hopeless to expect a

reduction of the mass of hypertrophied tissue by other methods. By the application of a 10 per cent. solution of cocaine, we not only render the parts insensitve, but are able to judge how much of the enlargement is due to fibro-plastic new growth which requires removal, and we are then in a position to determine what line of treatment to adopt.

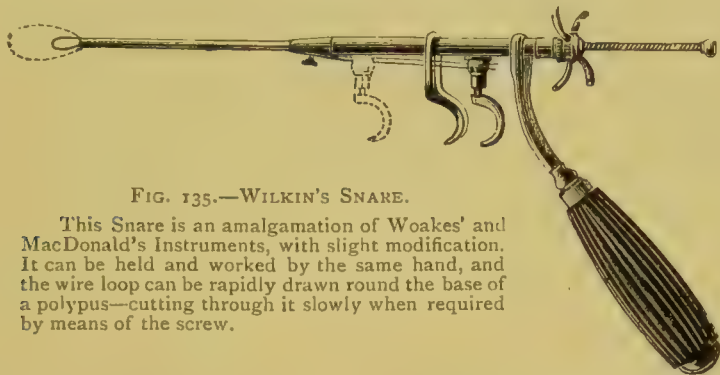


FIG. 135.—WILKIN'S SNARE.

This Snare is an amalgamation of Woakes' and MacDonald's Instruments, with slight modification. It can be held and worked by the same hand, and the wire loop can be rapidly drawn round the base of a polypus—cutting through it slowly when required by means of the screw.

Galvano-cauterisation.—In many cases it suffices to make one deep linear cauterisation along the whole length of the hypertrophied turbinal. Two or three of these cautery incisions may be necessary to reduce the hypertrophy, and at least one week should be allowed between each application.

Submucous incision, introduced by Delavan, has this advantage over the cautery, that it preserves the normal surface

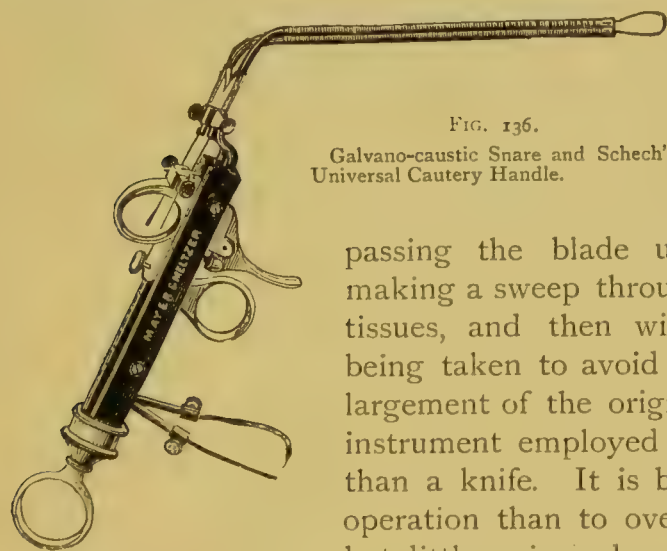


FIG. 136.
Galvano-caustic Snare and Schech's
Universal Cautery Handle.

of the mucosa. For this purpose use a small knife, first applying cocaine in the usual manner, and then

passing the blade under the mucosa, making a sweep through the sub-mucous tissues, and then withdrawing it, care being taken to avoid any additional enlargement of the original opening. The instrument employed is a needle rather than a knife. It is better to repeat the operation than to overdo it. There is but little pain and only slight bleeding.

It is a good plan to keep the cocaine in contact with the tissues for several hours, and to let the slight hæmorrhage stop of its

own accord. Delavan finds that this method is easy of execution and free from bad effects.

Snaring.—When the hypertrophy is large enough to secure with a snare, it may be removed by this means. The result obtained is highly satisfactory; and, though if the cold snare be very gradually tightened our object can be accomplished with little hæmorrhage, the galvano-caustic snare is preferable, as it cuts through the tissues more rapidly. It is often extremely difficult to engage these masses of hypertrophied turbinal tissue, and in many cases it is necessary to first transfix the mass with a Jarvis needle to prevent the wire of the snare slipping off as the loop is tightened.

Resection.—If the hypertrophy is very considerable, long curved scissors or Carmalt Jones' spokeshave may be cautiously used to remove, not the whole turbinal, but as much as is abnormal in its dimensions. Ablation of the whole turbinated body has been followed by severe hæmorrhage, and as an after result by collections of crusts in the nose, pharyngitis sicca, and laryngitis. Therefore, any operation involving resection of these important structures should be restricted to those cases in which less radical measures have failed, and only so much as is essential for the relief of nasal stenosis should be removed.

General Treatment is, however, quite as necessary in this affection as in simple chronic rhinitis, and all that has been said under this head in reference to simple chronic rhinitis applies equally to the more advanced hypertrophic form with this difference, that local treatment must occupy the first place in the latter disease, whereas it is comparatively unimportant compared with general therapeutics in the early simple rhinitis.

ATROPHIC RHINITIS.

Etiology and Pathology.—There is considerable diversity of opinion as to the pathology of atrophic rhinitis, and it must be confessed that the actual pathology of this common affection is at present an open question. Thus :—

(1.) Zaufal believes ozæna arises from a congenital deficiency of the turbinated bones resulting in undue patency of the nasal passages. It is difficult to accept such a view, for, though a disease of youth, it is never congenital, and, moreover, may undergo spontaneous cure.

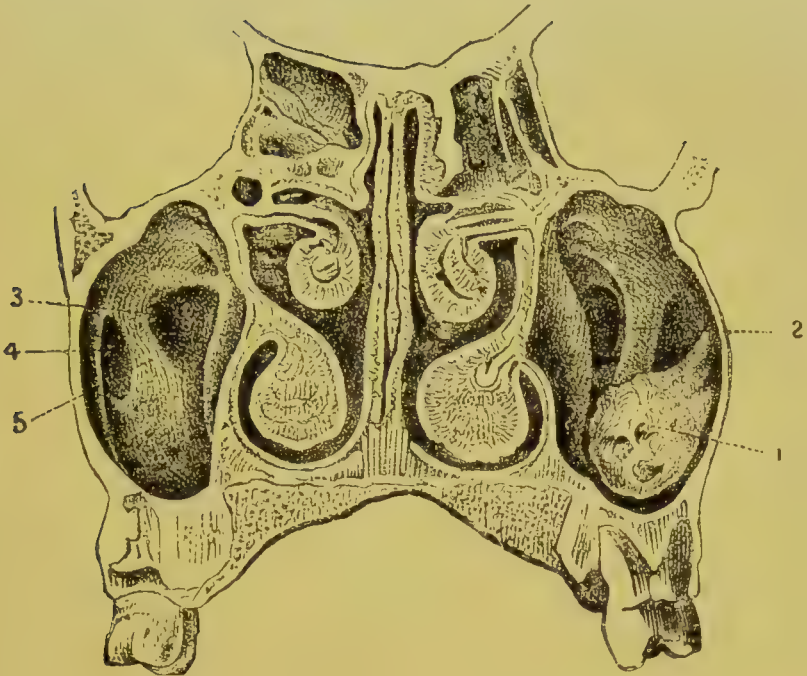


Fig. 1.

Transverse section through the nasal cavities and maxillary sinuses. The floor of the antrum is at a lower level than that of the nasal passages. A polypoid growth (1), and bony trabeculae (2), and other irregularities (3, 4 and 5) are seen in the cavities of the antra (Zuckerkindl).



Fig. 2.

Transverse section showing irregular development of the maxillary sinuses (Zuckerkindl).

(2.) The opposite view is held by Berliner, that it is associated with nasal obstruction, and due to pressure of the middle turbinal against the septum, with consequent defective secretion.

(3.) Habermann extending B. Fränkel's and Krause's observations, regards ozæna as due to fatty degeneration of the acinous glands and Bowman's glands, with inflammation and fibroid degeneration of the mucous membrane, resulting in atrophy.

(4.) Michel believes that accessory sinus disease, and Bosworth that purulent rhinitis of childhood, stand in causal relation to it.

(5.) The disease has been attributed to the action of many different micro-organisms. Vedova and Belfanti, in 120 cases, found the pseudo-diphtheria bacillus in all, and the bacillus mucosus in many. The pseudo-diphtheria bacillus has been found in a great many cases by other observers, and in several cases the true diphtheria bacillus. By Fränkel and Löwenberg, a coccus and a little bacillus (Pes-Gradenigo) have been held to be the active causative agent. Not one of the above organisms has been accepted generally as the specific organism of ozæna, but Löwenberg, Abel (100 cases), and Cozzolino (42 cases), found the bacillus mucosus present in every case examined. Cozzolino, in his 42 cases, found, in addition to the *b. mucosus*, the pseudo-diphtheria bacillus eight times, staphylococcus pyogenes aureus and albus nine times and seven times respectively, and numerous other organisms in a varying proportion of the cases. He considers that the *b. mucosus* is not the prime etiological factor in ozæna, but that it is responsible for the fœtor and formation of crusts.

(6.) In my opinion, the affection is of the nature of a tropho-neurosis, which, like acne vulgaris, is generally connected in some obscure manner with sexual development, or sexual involution at the time of the menopause.

It is often regarded as a final stage of chronic hypertrophic rhinitis. Undoubtedly, this is occasionally true, but in the great majority of cases the disease is primarily atrophic, and has been known to arise in infancy. *Ozæna* is a clinical symptom which is almost invariably associated with atrophic rhinitis, and is either due to some specific micro-organism, *e.g.*, the bacillus

fœtidus (Hajek), or is simply analogous to the peculiar odour of the secretion of the axilla or feet.

Though ozæna may be present without atrophy, it is never so persistent and marked as in atrophic rhinitis. From its constituting the most prominent symptom complained of in most cases of atrophic rhinitis, the symptom has given the name to the disease; but we may have, though rarely, atrophic rhinitis without ozæna, or ozæna without atrophy, just as we meet with bronchiectasis which may not be "fœtid," and "fœtid" bronchitis without symptoms of bronchial dilatation. Moreover, the disease may be unilateral, and is often more pronounced on one side than the other.

Wyatt Wingrave directs attention to the remarkable atrophy of the lymphoid tissue in the nasal and naso-pharyngeal tissues, and in fifty-six out of sixty cases found complete atrophy of the faucial and pharyngeal tonsils. He has also demonstrated that the sticky secretions and crusts are not muco-pus, for the elements of pus are wanting. The secretion is mucus with epithelial cells, multi-nucleated lymphocytes, with some staphylococci and other bacteria.

Though heredity appears to have some influence, and it is often associated with anemia and the strumous diathesis, ozæna is essentially a disease of puberty and young adult life, and the majority of cases are found in females.

Symptoms.—The patient usually complains of no pain, but of dryness of the nasal passages and naso-pharynx, alternating with profuse discharge when the masses of inspissated secretion come away; but the characteristic and intensely foul odour is not perceived by the patient, who has generally more or less completely lost the sensation of smell. The nose is often flattened and broad, with sunken bridge. Pain at the back of the eyes, or over the bridge of the nose, is sometimes present.

On *inspection*, the most noticeable features are the width of the nasal passages from atrophy of the mucous membrane, so that if the copious sticky secretion, which hangs in strings across the passages or collects in greenish grey masses, is removed, we can often see the pharyngeal wall. Collections of dry secretion are found on the posterior pharyngeal wall, and on examining the larynx and trachea we often find them similarly affected, and that collections of dark green secretion occupy the trachea, the

upper surface of the cords, or stretch across the glottis, causing more or less complete hoarseness or aphonia. The view that these are simply collections of inspissated secretion, which have fallen into the glottis from the naso-pharynx, is probably incorrect. When the nose alone is affected, the breath from the mouth will not have the characteristic odour. The atrophic process may extend also to the Eustachian tubes.

The disease is a source of great annoyance, but is never fatal, nor does it involve any great danger to the general health. It tends to undergo spontaneous cure in many cases about middle life, and especially in women at the conclusion of the menopause.

Diagnosis.—The bony structures never become necrosed, and the absence of any dead bone or perforation of the bony septum are points which enable one to differentiate between true ozæna and tertiary syphilitic disease, attended with accumulation of *mucopurulent* secretion, and which often closely simulates ozæna in the intense sickly odour which accompanies this necrotic process.

Atrophic rhinitis in infants and young children is very rare, and the possibility of its being due to inherited syphilis should be remembered (see p. 298).

Treatment.—In this, as in all forms of rhinitis, a general tonic and hygienic treatment must be adopted whenever there is any indication of impaired health. But ozæna is very commonly associated with apparently perfect general health.

In the *local* treatment, which often at best is only palliative, the first essential point is the removal of the crusts of inspissated secretion. This, to be effectual, requires patience and care, but is readily accomplished by projecting a stream of a simple alkaline wash on to the crusts at considerable pressure. The spray should be directed by the eye, with the nasal speculum *in situ*, and with the aid of a good illumination, and should be continued till every particle of secretion has been washed off. Large dry crusts may be gently removed by forceps, or by a probe covered with cotton wool. Similarly, the post-nasal douche must be used to get rid of all crusts in the naso-pharynx.



FIG. 137.
Collections of greenish inspissated secretions in Laryngo-tracheal Ozæna.

The best lotion to use is either simple warm water with a little bicarbonate of soda or sanitas added, or Dobell's solution.

The patient must be directed to use an alkaline nasal douche daily to prevent the accumulation of the sticky mucus and the formation of crusts. Warm water at 90° F., containing 1 or 2 per cent. of bicarbonate of soda and common salt, answers admirably for cleansing.

A simple hand-ball douche answers well, but the patient must be directed to use no force, to inject up the most blocked nostril, allowing the fluid to escape by the other side, and to be careful



FIG. 138.

Nasal Douche.

to have the lotion at the proper temperature (about 95° F.). Without great care there is always a risk of setting up *otitis media*, and for this reason the douche should only be ordered for cases like *ozæna*, and should be followed by a soothing, oily, antiseptic spray, or, as Christopher Heath recommends, a snuff composed of iodol and borax (1 to 7). Leffert's coarse nasal spray is very useful and sometimes more successful in removing crusts, while it is devoid of danger.

I have used, with some success, the daily insufflation of equal parts of citric acid and sugar of milk, as suggested by Hamm, the passage being first thoroughly cleansed by the douche; it seems to lessen the odour of the crusts.

The second point to consider is the best methods of increasing the functional activity of the atrophied mucous membrane, and these may be divided into (a,) excitation by electrical currents and (b,) stimulation by local applications.

Bryson Delavan states that the secretions are rendered more abundant by the application of the negative pole of the galvanic current to the affected parts.

Cupric Electrolysis.—This method, trying as it is to the patient, has yielded better results than any other with which I am acquainted, and though my own cases are too few and too recent to warrant any positive statement on the permanence of the

improvement it induces, I have been much impressed with the early beneficial effect of this method. McBride, in eight unselected cases, obtained a practical cure in four (lasting at least eighteen months). Hecht reports two cases, both receiving six applications, lasting for ten minutes, of from 25 to 30 milliamperes; in both cases there was improvement. The method is applied as follows: Having applied cocaine, or under chloroform, a copper needle attached to the positive pole is inserted into the tissues of the inferior or middle turbinated body, and a steel needle, attached to the negative pole, into the septum, and a current of from 5 to 30 milliamperes is passed from ten to fifteen minutes. The needles are then withdrawn. The stronger currents are rather painful, and therefore it is generally more convenient to use a current of from 10 to 15 milliamperes instead of putting the patient under chloroform anæsthesia. The procedure should be repeated at intervals of two or three weeks until the symptoms are in abeyance. The process should be repeated once or twice a year in cases which relapse. The favourable results noted are increased secretion and general vascularity of the mucous membrane, and diminution of fœtor and secretion. Logan Turner states that the same organisms are found in the nose after cupric electrolysis as before.

Injections of Diphtheritic Antitoxin were introduced by Vedova and Belfanti on the assumption that the disease was due to the presence of attenuated diphtheria bacilli. The method has been tried by a large number of practitioners, and in many cases temporary benefit has resulted. I have tried it in one case only, but the result was absolutely *nil*. It is said, however, to have a good effect in those cases in which cultures yield a predominance of pseudo-diphtheria bacilli. The bacilli do not seem to disappear, and therefore it is probable that any good effect is chiefly due to the stimulating effect of the serum on the nasal mucosa.

Galvano-cauterisation of the nasal mucosa is generally to be deprecated inasmuch as any destructive procedure, although yielding temporary benefit owing to the inflammatory reaction induced, is sure to increase the atrophic process. At most a very superficial cauterisation is permissible in certain cases.

Spraying or painting the nasal passages with stimulating antiseptic solutions, such as formalin, has been advocated by Bronner and by Richards. Richards, after cleansing the passages

and applying cocaine, uses a solution of formalin containing 5 to 10 drops of the 40 per cent. solution in 8 ounces of warm water; and one drop is added to the solution, which the patient uses in the douche cup for the daily cleansing. In bad cases Bronner prescribes a 1 in 1000 to 1 in 2000 solution of the liquid formalin with water, to be used with a small nasal syringe; or a 1 in 500 to 1 in 1000 solution with a little glycerin added, to be used with a coarse spray three or four times daily for a few days, and then two or three days a week for a few weeks or months. If the application is painful the solution should be further diluted. My experience of formalin solutions is that they are painful if strong enough to be effectual, and that they offer no advantage over the milder antiseptic and alkaline solutions recommended above.

Freudenthal claims excellent results from internal vibratory massage of the nasal passages. Gottstein's method of plugging the nares with medicated cotton wool, *e.g.*, cyanide of mercury gauze worn for one hour every morning, is fairly effectual but tedious and transient in its results.

MEMBRANOUS RHINITIS.

(Rhinal diphtheria and fibrinous rhinitis.)

Just as angina and laryngitis with membranous exudation are divided into two groups, the diphtheritic and non-diphtheritic, so must we separate into two classes cases of membranous or fibrinous rhinitis, *viz.*, (1,) rhinal diphtheria (see page 138) and (2,) fibrinous or plastic rhinitis.

FIBRINOUS OR CROUPOUS RHINITIS.

NON-DIPHTHERITIC MEMBRANOUS RHINITIS.

Definition —Fibrinous rhinitis, first described by Schüller, is an acute or chronic inflammation of the nasal mucous membrane characterised by the presence of false membrane, and due to the presence of the staphylococcus pyogenes aureus or streptococcus pyogenes, and though clinically these cases may be indistinguishable from the others, we are bound to separate them from diphtheritic rhinitis into a special class for the reasons already mentioned.

True (non-diphtheritic) fibrinous rhinitis is certainly less common than diphtheritic rhinitis. The Klebs-Loeffler bacillus

was found in no less than sixty-nine of Wishart's ninety-eight collected cases. The only means of distinguishing the affections is by bacteriological examination.

Symptoms.—Generally the only notable symptom is mucous discharge and obstruction of the nasal passage by false membrane of greyish-white colour, there being no false membrane in the fauces; the glands of the neck are not enlarged, the urine is non-albuminous, and there are no paralytic sequelæ. Sometimes the attack is ushered in with chilliness and rise of temperature.

The affection tends to be protracted, and the membrane re-forms several times after removal.

Burn Murdoch has reported a case in which the attacks recurred six times, at intervals varying from one month to a year, each attack lasting about a week or a fortnight. The first attack began in November with symptoms of an ordinary cold. In a few days the nose was completely blocked: there was a copious muco-gelatinous secretion and numerous fibrinous casts were shed, the nose and face being much swollen and painful, but in this case there was no rise of temperature. The subsequent attacks varied in severity. There were no paralytic sequelæ. W. F. Robertson's examination of the membranous casts of this unique case showed that they were composed mainly of fibrin containing numerous round cells. There were some epithelial cells suggesting shedding of the whole depth of mucous membrane. Sections stained by Loeffler's method showed no micro-organisms, and Gram's method only revealed a few groups of micrococci.

The majority of cases of so-called fibrinous rhinitis are really diphtheria, and cultures should invariably be made in order to avoid mistakes in diagnosis. If bacteriological investigation excludes diphtheria the diagnosis presents no difficulty.

Treatment consists in treating the general symptoms and improving the general health, and locally in the removal of the false membrane by forceps, and applying simple astringent and antiseptic aqueous solutions.

RHINITIS CASEOSA.

Rhinitis caseosa (*coryza caseosa*) originally described by Duplay, consists in a blocking of the upper regions of the nasal fossæ with caseous matter similar to that found in some

sebaceous cysts. The cheesy matter, which has an intensely foul odour, may form in considerable quantity, even causing facial deformity, and may lead to anosmia. In several cases the antrum or some other accessory sinus has been implicated, and it is probable that some cases at any rate are due to a collection of inspissated pus in one of the accessory cavities, finding an exit into the middle meatus or olfactory fissure. Massei expresses his conviction that caseous rhinitis is produced by the streptothrix alba which Guarnania has several times identified in true cases of caseous rhinitis.

The mucous membrane in the neighbourhood of the cheesy mass may be altered, resulting in the formation of highly vascular polypoid mucous membrane which bleeds readily and freely on the merest touch with a probe (Alexander).

The affection appears to be always unilateral, and the diagnosis depends on the presence of the foul caseating matter in the middle meatus or olfactory fissure.

The treatment consists in the removal of the mass, thorough cleaning of the region involved, and the usual treatment of any accessory sinus that may be involved.

RHINOLITHS.

Any foreign body lying in the nasal passages for a lengthy period is liable to become encrusted with calcareous matter and form a rhinolith. The presence of micro-organisms appears to be one factor in their causation, by attracting the lime-salts of the nasal secretion. In many rhinoliths no definite nucleus can be found, but in these it has usually formed round a nucleus of blood or mucus. Women seem more liable than men to develop rhinoliths; thus Seeligmann, in a collection of one hundred and ten cases, found that sixty-two were in female patients and twenty-nine in male, the sex not being recorded in nine.

The symptoms and treatment are practically the same as in other foreign bodies. They sometimes reach an enormous size, especially in tropical climates; thus Headley reports one which weighed 720 grains. They consist chiefly of carbonate and phosphate of lime, with about 30 per cent. of organic matter. The colour of a rhinolith varies from a dirty grey to brown or black.

XANTHOSIS.

A dark yellowish pigmentation of the pituitary membrane, a result of interstitial capillary hæmorrhage into the mucosa, has been described by Zuckerkandl under the term *Xanthosis*. It occurs either diffused over a considerable area, or in patches, especially in the anterior or cartilaginous portion of the nasal passages.

The affected mucous membrane is often more or less atrophied, and the cartilaginous septum may become the seat of ulceration or perforation as a consequence.

When an ulcer forms it should be treated by applications of protargol or some other suitable germicide, so as to obviate the ulceration spreading or going on to septal perforation.

CHAPTER XIII.

CHRONIC INFECTIVE DISEASES OF THE NOSE.

SYPHILIS—TUBERCULOSIS—LUPUS—GLANDERS—RHINOSCLEROMA—
HENPUYE.

SYPHILIS OF THE NOSE.**INHERITED SYPHILIS.**

THE *early form* occurs within the first three months of life, and assumes the form of a catarrh with tumefactions of the nasal mucosa with consequent "snuffles." Norval Pierce observes that syphilitic coryza differs from simple coryza in the infant in that the onset is less violent, is more gradual, and the secretion, especially in the first stage, is not so profuse, but when established lasts longer, and that disseminated patches of well-defined erythema may be seen, especially on the septum, on which may develop plaques, the first pathognomonic signs of syphilis. The discharge of mucus or muco-pus is irritating to the anterior nares, producing excoriations and fissures, and tending to form crusts in the nasal passages, which become somewhat fœtid. At this period necrosis of the cartilage is rare. When atrophic rhinitis attacks a child, and its bilateral appearance cannot be satisfactorily accounted for, the personal and family history should be investigated for indications of a congenital syphilitic taint.

The *late form* manifests itself between the age of five and puberty. It corresponds to the tertiary period in the acquired form, and is, therefore, characterised by gummy infiltration, caries, and necrosis of the cartilage of the septum, the vomer, and the turbinated bones, with fœtid discharge and with consequent deformities.

ACQUIRED SYPHILIS.

Primary sore is very rare. The most usual site is on the ala nasi; less frequently it occurs just within the nasal passage beyond

the vestibule. The chancre is generally due to inoculation with the finger nails, but kissing and other modes of infection are recorded. The chancre does not differ in aspect from chancre elsewhere, but the secretion from the resulting catarrh is apt to collect and become inspissated. In young children the sore may appear as a simple papule without induration (Massei). In adults it is usually attended with induration and often with much swelling of the nasal mucosa, and feverishness. The sub-maxillary and sublingual glands and those in front of the ear usually show marked indolent swelling.

Secondary syphilis gives rise to slight symptoms, chiefly nasal *catarrh* with tumefaction of the Schneiderian membrane. *Mucous patches* may occur in the vestibule or less frequently on the septum and inferior turbinated bodies, but they are much more rarely observed in the nose than in the mouth or pharynx. The patches tend to secrete freely and to undergo superficial ulceration.

Tertiary syphilis may assume the form of a localised gumma of the septum or turbinated bodies, a firm, circumscribed, red swelling. More generally we find extensive ulceration and suppuration, with caries or necrosis of the cartilaginous septum, the vomer, and the turbinated bodies. The discharge is considerable, purulent, bloody, yellowish-green, but is apt to collect and form foetid greyish-white or greenish-black crusts. If bony caries or necrosis has occurred—and it is but rarely that syphilitic ulceration is confined to the septal cartilage—the stench is indescribable, and most penetrating to those around, though to the patient the sense of smell is diminished or altogether lost: a probe will detect diseased bone concealed by the greenish necrotic tissue.

The disease is generally bilateral though often more advanced on one side. Syphilitic infiltration of the cartilage and bone may result in: (1,) necrosis, with exfoliation; (2,) rarefying osteitis; or, (3,) formative osteitis with periosteal or perichondral thickening. Pieces of necrosed bone may be separated from time to time with the discharge, and in consequence of the contraction of the subcutaneous connective tissue, or from the partial or complete destruction of the nasal bones and septum, the nose becomes characteristically broad and sunken, “saddle-backed,” or the whole of the cartilages and tissues of the alæ,

and even the whole external nose may be lost. Following gummy infiltration, the bone may undergo partial absorption without necrosis.

Various complications may arise from extension of the syphilitic infiltration to the sphenoid or ethmoid, such as meningitis, thrombosis of the cavernous sinus, or involvement of the optic nerves or those passing through the sphenomaxillary fissure, with symptoms similar to those complicating purulent inflammation of these sinuses.

Diagnosis.—The diagnosis should rarely present much difficulty. Syphilis may attack any part of the nose, but the loss of tissue is usually chiefly confined to the septum. The history and concomitant lesions will generally be enough to confirm the diagnosis which the nasal features suggest.

Syphilis in the nose must be differentiated from tubercle, lupus, malignant disease, and ozæna (see page 288). Tuberculous disease is most likely to be confused with syphilis of the nose, while the difficulties in diagnosis may be further increased by the association of syphilis with tubercle, lupus, or leprosy.

Treatment.—In addition to the general treatment appropriate to the particular phase of syphilis present, local treatment is usually necessary.

In young children at the breast, syphilitic catarrh by interfering with nasal respiration may render suckling impossible. The child should be fed by the spoon till the condition has yielded to the internal administration of mercury in small doses. If, as is often the case, the infant declines all nourishment from any but the natural source, we may spray a weak solution (2 per cent.) of cocaine, or a solution of menthol (20 per cent.) in olive oil or liquid vaseline into the nares before putting the child to the breast, as recommended by McBride.

The special treatment in syphilis of the nose consists in the use of alkaline and cleansing douches to keep the passages free from the accumulations of secretion. In tertiary ulceration insufflations of aristol or iodol tend to keep the fætor under, and calomel fumigations may be useful in checking the progress of the disease; but with necrosis of the bone it is useless to expect any means of overcoming the stench to succeed. The dead bone should be gently removed if possible.

The importance of prophylactic measures to prevent the possibility of others becoming infected is too obvious to require insistence here.

TUBERCULOSIS OF THE NOSE.

Etiology and Pathology.—Tuberculous disease confined to the nasal passages is somewhat rare. It more usually occurs in the course of pulmonary or laryngeal tuberculosis.

Weichselbaum in 146 autopsies on patients who had died with tuberculous disease, found only two cases in which the nose was implicated, and though I have only seen a few of such cases myself, it is probable that a systematic examination of the nose in cases of advanced phthisis would show that it was less rare than is supposed. M. Herzog, who has reported 10 cases of his own, and has collected and reviewed the literature of all the instances recorded, 80 in number, finds it occurs in the form of (1,) neoplasms; or (2,) ulcers; or (3,) a combination of both forms. In a collection of 90 cases by Heryng, ulcers were present in 48 cases, and in 42 tumours occurred.

(1,) *Tuberculous deposits* are found from the size of a poppy seed to a large walnut, although they are seldom larger than a split-pea. They are soft and friable, and bleed easily, of irregular outline, rounded or elliptical, of reddish or greyish-yellow colour, surrounded by elevated soft margins in which sometimes miliary tubercles are seen. This description applies to most of my cases, but I have seen a tuberculous growth on the septum which, in the early stages, was smooth and red, and well defined, resembling a small sarcoma or fibroma in appearance. Polyak also records a case of a tuberculous growth attached to the anterior portion of the septum, which on removal measured 30 m.m. long, and 40 m.m. thick. There was no tuberculosis detected elsewhere either in this or in my own patient.

(2,) *Tuberculous ulcers* here, as in the pharynx, are shallow, irregularly round or oval, with clean cut, or with soft, slightly elevated margin, and presenting a greyish-yellow base filled with caseous tubercles. They are usually situated on the cartilaginous

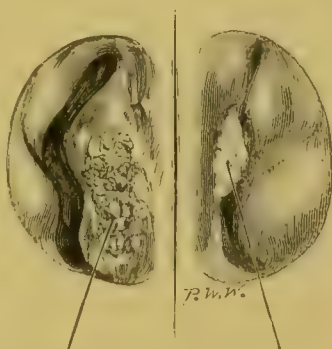


FIG. 139.
Tuberculous
neoplasm of the
septum.

FIG. 139a.
Sticky secretion
and early tuber-
culous deposit.

portion of the nasal septum, near the anterior border ; but they may extend to the ala nasi or even to the upper lip. The tubercular deposit, of course, consists of granulation tissue, with giant cells, and contains tubercle bacilli, which are most abundant in the deeper layers.

Infection probably takes place from inoculation by the finger-nail. Purdon reports a case of a healthy patient who infected her nose by frequently lending her handkerchief to a consumptive sister.

Symptoms.—At first the symptoms are very indefinite, but as the deposit increases nasal obstruction may occur. The small miliary tubercles very soon ulcerate, and then pain and a mucopurulent discharge comes on, often bloodstained, and after continuing a short time becoming very fetid. In fact, cases of tuberculous disease of the nose closely resemble ozæna, from which one may distinguish them by finding the characteristic bacillus. The absence of pain is remarkable.

Diagnosis. Tubercle may be differentiated from tertiary syphilis by the presence of tubercle in other parts, by the progressive character of the lesion in spite of anti-syphilitic treatment, and by the presence of the bacillus, and from its selecting the cartilaginous septum and not the osseous. It must also be distinguished from lupus, ozæna, fibroma, malignant disease, and chronic glanders.

Treatment.—The local treatment consists in thorough removal of the deposits by scraping, and the subsequent application of chromic acid, lactic acid, or the galvano-cautery, followed by some antiseptic insufflation daily, such as aristol, eucrophen, etc. Sometimes I have found fuming nitric acid the most useful application, followed by orthoform insufflations to obviate pain. The disease is extremely prone to recur after a short period.

Of course, the usual general treatment for tuberculosis should be pursued at the same time.

LUPUS OF THE NOSE.

Etiology and Pathology.—Intra-nasal lupus is extremely rare, unless we include those cases of lupus of the ala nasi which invade the vestibular portion.

The nodules occur generally as multiple, small, hard, elastic tubercles, covered with dry brown scales of inspissated secretion,

or greenish-yellow pus. Beneath the scab will be found the characteristic ulcers, round or oval, cup-like, with raised indurated margin, tending to cicatrise at one part and extend in another, and painful if pricked with a probe. They are generally situated on the septum, near the anterior inferior extremity, or close to the floor, and sometimes on the anterior part of the inferior or middle turbinals. When the crusts separate there is a certain amount of bleeding, followed by sero-purulent discharge which may be very offensive if the crusts have been long retained.

The bones are very rarely, if ever, involved in the necrotic process, but the cartilaginous septum generally becomes perforated sooner or later.

Symptoms are mainly those of nasal obstruction, with a limited amount of intermittent discharge. The patient usually complains of irritation in the nasal passage, but pain is generally absent or only slight. Other symptoms will be present, of course, if the pharynx and larynx are involved. In one of my cases epiphora was the only symptom noticed, due to obstruction of the lower end of the nasal duct.

Diagnosis is generally easy, inasmuch as intra-nasal lupus is usually associated with lupus of the skin or pharynx.

It is distinguished (1,) from syphilis by the destructive process not extending to the bony structures, the tendency to heal, and the slight amount of discharge, and the absence of that intensely sickening odour of syphilis, which is not removed by the most careful cleansing; (2,) from malignant disease by its slow progress and tendency to heal, and from epithelioma, though not from sarcoma, by its occurrence in the young; (3,) from nasal tuberculosis by the appearance of the deposit, and its slow course. But it is often impossible to differentiate between lupus and tuberculosis of the nose, and if the view that lupus is but a mitigated form of tubercle be correct, this resemblance can be readily understood.

For other points in the differential diagnosis, see "Lupus of the Pharynx and Larynx" (p. 145).

Treatment consists in removal of the crusts, and thorough cleansing of the passages, followed by curettement and the application of lactic acid, or strong nitric acid and other local agents, as in lupus of the pharynx. A small patch may be destroyed by the galvano-cautery.

Tuberculin in minute doses may be tried with advantage. I have had successful results in several cases of lupus. The dose of the injection should never be sufficient to cause very marked local reaction, and for this reason it is well to commence with $\frac{1}{1000}$ of a gramme of the new tuberculin of Koch, and gradually increase it.

Larger growths may be removed by the galvano-caustic snare, lactic or nitric acid being subsequently applied to the base.

General treatment by tonics, cod-liver oil, iodide of iron, arsenic, etc., must of course be combined with local treatment.

GLANDERS.

Etiology and Pathology.—Glanders is a contagious disease of horses and cattle, due to the specific micro-organism the *b. mallei*, but is occasionally met with in men, who generally contract it from infected horses, and thus it is usually seen in ostlers and grooms. Morell Mackenzie records a fatal case, which resulted from a diseased horse sneezing when being driven in a hansom, some of the secretion coming into the face of the patient who was inside.

It occurs in the acute and chronic forms. The disease may be chronic at the outset, subsequently passing into the acute variety.

Chronic glanders closely resembles tertiary syphilis of the nose, but it is extremely rare. The mucous membrane of the nasal fossæ is slightly swollen and may be painful, and is covered with dirty scabs. There is a peculiarly viscid, offensive, mucopurulent discharge, and when ulceration supervenes it is serous. The disease may extend to the pharynx, back of the tongue, and larynx.

A large proportion of cases end fatally in six to eight months. As the bones are often implicated in the ulcerative process, the diagnosis from syphilis is rendered all the more difficult, and often depends on the absence of any improvement from anti-syphilitic treatment.

Acute glanders is the commoner form of the disease in man, but, according to Böllinger, who collected 120 cases, it is less frequently localised in the nose, than is the case in the horse.

Symptoms.—The general symptoms are ushered in by a general febrile condition, with headaches, rigors, and pains of a

rheumatic character in the limbs. In a day or two a pustular nodular infiltration occurs in the nasal mucosa, with a profuse glairy discharge. The nodules ulcerate, and the discharge becomes more viscid and muco-purulent, while the nose externally becomes red, painful, and swollen.

A papular eruption resembling small-pox, comes out on the face and limbs; diarrhœa, profuse sweating, vomiting, and general prostration, usually end in coma and death in less than three weeks.

Diagnosis.—The general symptoms closely resemble acute rheumatism or typhoid fever at the onset, but the extremely adynamic condition should serve to exclude acute rheumatism and tertiary syphilis, while the absence of other features serve to eliminate typhoid fever and small-pox. From anthrax it would be distinguished by the absence of the "charbon pustule."

Treatment is practically hopeless, but should be conducted on general principles, for which the reader will consult works on general medicine.

Locally, Elliotson reported success in stopping the nasal discharge, by injecting a solution of 2 grains of creasote in a pint of water three times a day. Other strong antiseptic agents will suggest themselves.

It is hardly necessary to emphasise the extreme importance of the most rigid prophylactic measures to prevent the inoculation of healthy individuals.

RHINOSCLEROMA.

Etiology and Pathology.—The cause of this rare affection, originally described by Hebra, is uncertain, though Stepanow and Cornil have described bacilli which they claim as the specific infecting agent. Shattock, in the Royal College of Surgeons' museum, has figured a streak culture of the bacillus grown on agar and stained with carbol-fuchsin. The micro-organisms consisted of spherical elements occurring singly, but most frequently in pairs or short chains, and rod forms may also be met with. The diplococci have been likened to Friedländer's pneumo-coccus. Lemeke holds that rhinoscleroma is identical with Störk's blennorrhœa, and the chronic subglottic thickening known as chorditis hypertrophica inferior, which follows Störk's blennorrhœa, is considered to be identical with rhinoscleroma.

It generally commences in the vestibulum nasi in the form of small, hard, raised nodules, which very gradually spread to the nasal passages. The nodules are hard, tender on pressure, covered by normal skin, and non-inflammatory. It may begin in this manner in the nose, or may take the form, *ab initio*, of diffuse thickening of the mucosa without showing any nodules. The mucous membrane is normal or slightly reddened in colour, smooth and shining, and very hard. The infiltration consists of small round cells which subsequently become spindle-shaped. There is no inflammation, no discharge or pain, and no ulceration. It may spread to the pharynx or the larynx, or even to the trachea, or the disease may commence in the pharynx or larynx without involvement of the nose. It generally occurs in advanced adult life, but Semon's case was in a boy of 14, a Guatemalan. Very few cases are recorded as having occurred in England, but it seems fairly prevalent in Austria and in some parts of Egypt, South America, and India.

Symptoms are entirely local, viz., stiffness and nasal obstruction, and the course of this disease is very chronic. The nose is peculiarly broadened and the tip approaches the upper lip, so that the nose looks flattened. The skin becomes indurated and knobbed. The nasal passages are narrowed by the very hard, smooth nodules and by the contraction of the infiltrated mucous membrane. It generally spreads gradually back, and if the pharynx be involved, it creeps down the posterior surface of the soft palate, to the palatal pillars and posterior wall. When the larynx is the seat of infection, it is the subglottic portion which generally suffers most, so that greyish-red nodular tumours may be seen projecting inwards. The epiglottis may be attacked. The infiltrated tissues undergo contraction, somewhat like the cicatricial contraction of lupus or the adhesive form of syphilitic infiltration. Consequently the nose becomes stenosed, the soft palate peculiarly wrinkled, and ultimately the contraction of the palate and pharyngeal wall may cause a ring-like narrowing similar to the adhesion of the soft palate to the pharyngeal wall following syphilitic ulceration. The contraction of the subglottic infiltration causes the glottic aperture to assume a circular form, even if the cords themselves are not involved in the disease. The main symptoms, then, will be those due to obstruction to respiration.

The **Diagnosis** is not always easy in the earlier stages, but the ivory-like hardness of the nodules and the absence of ulceration will form some guide, while the more advanced cases are almost unmistakable. Unlike lupus or syphilis, there is no ulceration. The affection is most likely to be confused with either lupus or syphilis, and in doubtful cases the patient should always be put through a course of anti-syphilitic treatment. The diagnosis should, however, depend on cultures made by inoculating blood serum with a needle that has been plunged into the affected tissues, or from the blood issuing from the pricked spot.

Treatment.—The mechanical obstruction may be reduced by the galvano-cautery or knife. No treatment hitherto introduced appeared to be of the slightest use in arresting the disease, till Stoukovenkow, of Kiew, had successful results in one case with interstitial injections of Fowler's solution of arsenic (1 to 12 per cent.). The treatment was continued for fifteen months, and no less than 222 injections were made.

Freudenthal prepared rhinosclerine for the treatment of his case, acting on the results of Pawlowsky's researches, which showed that by these injections good results may be obtained.

An article on "Rhinoscleroma," by Freudenthal (New York Med. Journ., Feb. 1, 1896), gives directions for the preparation of rhinosclerine, and includes an excellent description of the disease, to which I am much indebted.

HENPUYE.

Henpuye, or "dog-nose," is an affection causing a characteristic external deformity of the nose, which is practically confined to the natives of the Gold Coast of West Africa and the West Indies. Chalmers, from personal observation of numerous cases, states that henpuye starts as a small bony swelling symmetrically placed on either side of the nose, during or soon after an attack of yaws in which there is a history of the nasal mucous membrane being attacked. This swelling, generally oval with the long axis directed downwards and outwards, is attached to the nasal bones, the nasal processes of the superior maxillæ. It is produced by the deposition of cancellous bone under the periosteum. The growth is generally bilaterally symmetrical. The symptoms are pain in the nose, and later headache and pain in the swelling during hot weather. Apparently the only effective method of treatment is removal by the saw and bone forceps.

CHAPTER XIV.

DISEASES OF THE PHARYNGEAL TONSIL.

HYPERTROPHY OR POST-NASAL ADENOIDS—CONGENITAL RHINO-PHARYNGEAL STENOSIS—RHINO-PHARYNGITIS.

THE collection of lymph follicles on the roof and posterior wall of the rhino-pharynx, known as the pharyngeal or Luschka's tonsil, is similar in structure and liable to the same diseases as the faucial and lingual tonsils. It may be the seat of acute inflammation, mycosis, syphilis, tubercle, etc., all of which are described in connection with these affections of the tonsil and pharynx. But it very frequently becomes hypertrophic, and, in many cases, the adenoid hypertrophy is considerable, forming "post-nasal growths" which give rise to special and characteristic symptoms, the clinical importance of which, first emphasised by W. Meyer, in 1868, is now widely recognised by practitioners.

HYPERTROPHY OF THE PHARYNGEAL TONSIL.*POST-NASAL ADENOIDS.*

Etiology.—The pharyngeal tonsil is normally present at birth, and remains well developed up to the time of puberty, at which time it should begin to atrophy; so that at the age of about eighteen or twenty its dimensions are insignificant or it has wholly disappeared. The lymphoid tissue may be unduly developed at birth and, as in the earlier years of life all lymphatic structures are especially active and respond to various incidental exciting causes of lymphoid hypertrophy, the symptoms generally date from birth or early infancy, and become well marked as the hypertrophy increases, by the fourth or fifth year, if not before. The majority of cases come under notice between the ages of five and fifteen, for although the hypertrophy often persists in adult life, it usually participates in the retrogressive changes

PLATE XXII.

Fig. 1.

The typical facial aspect due to post-nasal adenoids.



Fig. 2.

To show the V-shaped, vaulted hard palate, associated with buccal respiration, due to "adenoids."



that normally occur at puberty, while increasing roominess in the rhino-pharyngeal space also diminishes the obstruction to respiration. All conditions which result in hypertrophy of the faucial tonsils, *e.g.*, the strumous diathesis and various exanthems, are likewise causes of post-nasal adenoid hypertrophy, and thus post-nasal growths and large faucial tonsils very frequently co-exist. Not infrequently the symptoms date from an attack of measles, scarlet fever or influenza. The influence of *heredity* is seen in many families, for it is by no means unusual for several brothers and sisters to suffer, and often enough the parents bear ample evidence in their faces of the mischief done by adenoid growths in their childhood, which persists throughout life, although the growths themselves may have long disappeared. Other evidences of inherited developmental defects are often found associated with the post-nasal affection.

Climatic conditions undoubtedly influence the occurrence of the growths, which are more frequent in cold and damp climates such as Holland, than in warm and dry countries such as Italy, where, according to Massei, the disease is but rarely observed in marked degree.

Bobone, of San Remo, even considers that pure and simple involution of adenoid growths is possible in the Riviera owing to the dryness of the climate, which also accounts for the extreme rarity of the vegetations amongst the natives there.

A tendency to catarrhal affections of the whole respiratory tract is almost constant in patients with adenoid growths, and this often results in intra-nasal obstruction. It is often stated that nasal obstruction is a frequent cause of post-nasal growths, but though it may have some influence, I am convinced that it is quite a minor factor, and that its influence is sometimes unduly exaggerated.

Pathology.—The growths are made up of lymph follicles, *viz.*, a reticulum filled with lymphoid cells, the trabeculae consisting of branching connective tissue cells, together with vessels and nerves. They are thus similar in structure to the enlarged faucial tonsils excepting that there is much less connective tissue element, they are more vascular and soft, and the surface is covered with ciliated epithelium. Rarely they are tuberculous, and in a few instances they contain cysts.

The growths occupy the roof and posterior wall of the rhinopharynx; in very young children they are more developed on the roof, but if developed towards puberty they are often confined to the posterior wall (see *Fig.* 140). Usually the hypertrophy



FIG. 140.

Adenoids. Section through the post-nasal region in adolescence, showing hypertrophy of the pharyngeal tonsil. ZUCKERKANDL.

either forms a large central cushion-like mass, or is disposed in longitudinal ridges with intervening furrows which, being viewed by posterior rhinoscopy "end on," have the appearance of an irregular row of mammillar or pear-shaped projections. But in many cases the disposition of the lymphoid hypertrophy is much more diffuse, and may occupy the whole of the roof and upper part of the posterior and lateral pharyngeal walls, including the fossæ of Rosenmüller, and even the lips of the Eustachian tube and the salpingo-pharyngeal fold may be quite prominent and nodular in appearance from lymph-follicular hypertrophy. In other cases bridges of lymphoid tissue will form, connecting the Eustachian lips with the hypertrophied

mass on the posterior wall. This diffuse form of hypertrophy may cause definite symptoms, although not blocking the posterior nares.

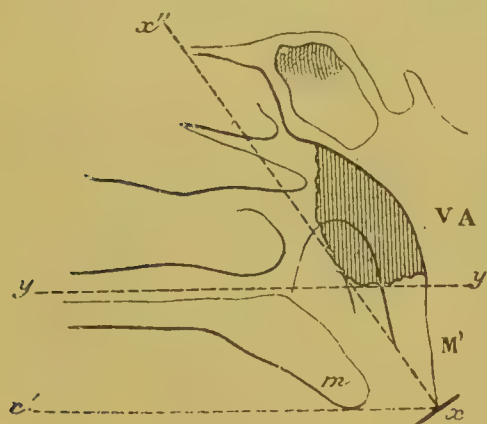


FIG. 141.

Diagrams of sagittal section of a rhino-pharynx with post-nasal adenoids after ZARNIKOJ. (*V.A.*) Adenoids, the lower margin of which reaches to level of *yy*, though on rhinoscopic examination, being viewed in the direction *xx''*, they appear foreshortened.

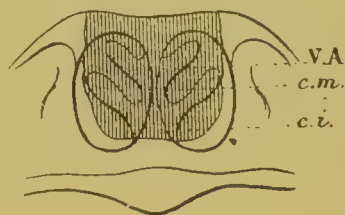


FIG. 142.

Actual vertical dimensions of the adenoids in *Fig. 141* after ZARNIKOJ.



FIG. 143.

Dimensions of the adenoids in *Fig. 141* as they appear in rhinoscopy after ZARNIKOJ.

Symptoms.—There are no absolutely pathognomonic symptoms of adenoid growths, yet although the indications of their existence vary greatly in degree according to the position and amount of hypertrophy, they are generally sufficiently characteristic for the practised eye to make a diagnosis from the physical appearance and facial aspect alone. The nose, small and undeveloped, becomes pinched, the alæ nasi fall in from long-continued disuse, a dimple appearing between the superior and inferior lateral cartilages. The upper jaw is narrowed and the hard palate vaulted and V-shaped; and as the upper lip is short and retracted, it often fails to cover the prominent upper incisors. The naso-labial fold is more or less obliterated, the inner canthus of the eye is drawn down causing drooping of the upper lid and a drowsy aspect. The open mouth and dropped jaw give an elongated, narrow appearance to the face (see *Plate XXII.*)

Respiration is peculiarly noisy and snuffing, especially noticeable during eating and drinking and during sleep. In the day-time, though the nasal passages are seldom completely

blocked, respiration is chiefly conducted through the mouth. Buccal respiration and breathing are particularly noisy at night, and suffocative night-terrors often occur, because during sleep the physiological nasal respiration re-asserts itself and thus the breath is drawn less through the mouth and more through the obstructed nose, with snuffling and snoring and partial asphyxia until the child wakes.



FIGS. 144 and 145.

Deformity of chest due to adenoid growths in early boyhood.

The habit of breathing through the mouth and persistent respiratory obstruction leads to frequently-recurring colds and bronchial attacks, peculiar chest deformities, and imperfect æration of the blood, conditions which result in defective growth and a general anæmic and weakly state of health. Symmetrical retraction of the cartilages of the lower ribs and ensiform cartilage, causing a keel-like prominence of the sternum (pigeon-breast) and infra-mammillary depressions, are very often due to persistent respiratory obstruction during childhood, when the chest-walls are soft and yielding, and in the majority of cases post-nasal growths are the source of the obstruction. The portion of the lungs corresponding to the retracted areas in the chest-wall are more or less collapsed. Moreover, as Eustace Smith has pointed out, the upper parts of the lungs are often

found collapsed in very young children with adenoids. The patch of collapsed lung here produces dulness on percussion and weak, harsh breathing in the supra-spinous fossa; sometimes very hollow breath sounds, conducted from the obstructed pharynx to the upper part of the chest on either side, leads to a mistaken diagnosis of grave pulmonary affections.

A peculiar, harsh, bronchial cough, bronchitis, and other pulmonary troubles are very commonly caused by post-nasal growths.

Speech is nasal and toneless, while stuttering and stammering are sometimes due to post-nasal growths.

Deafness.—The ears should always be examined. From extension of the pharyngeal catarrh, or by direct pressure of exuberant vegetations, the orifices of the Eustachian tubes become blocked, and then from the gradual absorption of the

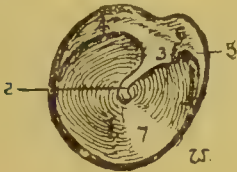


FIG. 146.

The normal right membrana tympani :—
(1) The membrane; (2) The handle of the malleus, ending in the umbo; (3) Short process; (4) The posterior, and (5) The anterior fold; (6) Membrana flaccida; 7) The bright spot.



FIG. 147.

The appearance presented by a depressed (right) membrana tympani. The handle of the malleus is foreshortened, and the short process, as well as the posterior fold, is abnormally prominent. The bright spot is more diffuse and less distinct towards the periphery of the membrane.

air in the middle ear the tympanic membranes often become very much depressed, or from *otitis media purulenta*, perforation of the drumhead may result.

Partly to the deafness, though largely to the deficient aëration of the blood and general impairment of health, are due the dulness, headaches, giddiness, and inability to fix the attention (Guye's *aproxesia*), that are such constant features in children with adenoids.

Many neuroses have been associated with adenoids, nor can it be a matter for wonder that lacking in stamina, stunted in growth, and defective in nutrition—as children with adenoids almost always are—they should exhibit marked instability of the cerebro-spinal nervous system. The co-existing nervous

phenomena and adenoids are doubtless only indirectly interdependent in most cases, yet in a few cases it is permissible to regard the nervous disturbances as reflex neuroses; thus such affections as asthma, stammering and stuttering, laryngeal spasm, nocturnal enuresis, convulsions, and even epilepsy, have been known to disappear shortly after the removal of the growths. The intimate association between the upper and lower respiratory tracts is well shown by the frequently observed fact that inhibition of respiration may occur on introducing the finger or forceps into the rhino-pharynx in operating, and probably the intensely disagreeable sensation of choking produced by digital exploration for adenoids is largely owing to the same cause. The constant irritation due to the presence of the growths, coupled with the pernicious effect of mouth breathing on the bronchial mucosa, affords a ready explanation of the asthmatic phenomena.

Soft adenoids bleed very readily, and thus considerable quantities of blood may be coughed up, or pass into the stomach and be vomited.

Examination may be made either by posterior rhinoscopy, or by palpation with the finger. It is of great assistance in subsequent operative treatment to obtain a good view of the extent and location of the growths, and, except in very young children, I rarely have much difficulty in seeing all that is necessary.

If the posterior nares can be inspected, we find either a group of pinkish gray gelatinous-looking masses, or one large mass with irregular surface, growing from the roof and posterior wall of the naso-pharynx (see *Plate XXVIII, Fig. 3*). With small growths, only a part of the post-nasal space is blocked up, but the amount of growth varies greatly. Very frequently the orifices of the Eustachian tubes and the upper part of the post-nasal apertures are concealed by their presence, or bridges of adenoid tissue may have formed between the lip of the Eustachian tube and the posterior pharyngeal wall, stretching across the fossa of Rosenmüller.

Palpation alone must often be relied on for the purpose of diagnosis in children, the right fore-finger, protected either by a finger-guard, or by a cork or napkin held between the teeth, being quickly passed up behind the soft palate and swept over the whole post-nasal space so as to detect the presence, extent and situation of any adenoids. The tip of the right finger

"Koplik's Spots" and Diseases of the Nose.



FIG. 1.



FIG. 2.



FIG. 3.

FIG. 1.—"Koplik's Spots" the pre-exanthematous sign of morbilli. The red spots of the buccal mucosa have coalesced, forming a mottled hyperemic area, on which numerous minute bluish-white specks are visible.

FIG. 2.—Vascular engorgement of the inferior turbinated bodies.

FIG. 3.—The posterior rhinoscopic image in an adult showing adenoid growths in the vault of the rhinopharynx, and hypertrophy of the inferior turbinated body.

should be passed *first behind the right posterior palatine arch and then up* to the roof of the pharynx, otherwise if the finger is inserted in the median line the palate is firmly retracted against the posterior pharyngeal wall, so that the finger cannot be made to pass up into the post-nasal space. In children adenoids are gelatinous, soft, and pliable, and readily bleed, but they often have a firm fibrous base, while in adults the hypertrophy of the pharyngeal tonsil is deeper red in colour, firmer in texture, and less exuberant.

Attention should be directed to the condition of the faucial tonsils, and of the lymphatic glands in the neck, which are often enlarged.

Diagnosis.—In children the facial appearance already described is almost pathognomonic, nevertheless all the symptoms of adenoids may be due to nasal stenosis combined with defective mental development, and it is well to avoid too hasty a diagnosis until the post-nasal space has been either inspected or felt, and the diagnosis placed beyond dispute. But the result of digital exploration may be misleading in several ways: *Firstly*, the practitioner may not realise that in young children a pharyngeal tonsil is a normal structure and will readily bleed on palpation; the mere presence of some soft adenoid tissue which can be felt, or of blood on the exploring finger does not warrant a diagnosis of post-nasal growths in the absence of characteristic symptoms. *Secondly*, in older children the finger may be directed only to the front part of the roof which may be free, while abundance of growth may lie undetected on the posterior wall of the space. *Thirdly*, there may be no large mass, the hypertrophy being diffuse, or occupying the fossæ of Rosenmüller only, and thus may escape notice. *Fourthly*, enlargements of the posterior turbinal bodies may not only cause the symptoms of nasal obstruction but may be mistaken for adenoids by a careless or unskilful examiner.

The symptoms are not all due or proportionate to the amount of nasal obstruction, and the absence of mechanical obstruction to respiration cannot be held to exclude adenoid hypertrophy which may be causing very pronounced symptoms calling for treatment, especially when the ears chiefly bear the brunt.

Prognosis.—The prospects of the patient depend on the amount of hypertrophy, on the existence of complications, on the

age at which the symptoms appear, and, above all, on operative treatment. The younger the patient the greater the risk of chest deformity, deafness and permanent impairment of health ; but, on the other hand, the most brilliant results of operation are obtained in young patients, because they have time to outgrow the effects of the disease before the period of physical and mental development has passed never to return. In any case, provided operation be not delayed until the deafness, chest deformity, facial defects, and other complications have become permanently established, the prognosis is uniformly most gratifying. The anæmic, pasty, puny, listless, dull woe-begone child becomes rosy, strong, bright and intelligent, and instead of snuffling respiration, open mouth, and constant catarrhal and bronchitic attacks, the lungs are properly filled and respiration normally conducted through the nose. Three questions are often put by the parents : " Won't the child grow out of the affection spontaneously ? " " Is an operation necessary ? " and " Will it afford a permanent cure ? " Our reply should be as follows : If the child be under twelve years of age, while conceding that it is not unusual for the growths to atrophy spontaneously about puberty, and that the child may escape permanent injury, yet the patient ought not to be subjected to the constant ill-health and serious risk of serious complications when the defect can be overcome by an operation which, if skilfully done, is practically free from risk and almost certain to be entirely successful. We cannot definitely promise that the growths will not recur, for even after very thorough and complete extirpation, they do return after a variable interval in a few cases, especially in young children.

If the patient is over the age of twelve, and no complications have arisen, we may delay operation in the less marked cases in the hope that the growths will disappear at puberty.

Treatment.—Before referring to the treatment of post-nasal adenoids, it may be well to direct attention to the extreme importance of dealing with all the associated conditions, or those which stand in causal relation to the naso-pharyngeal growth : firstly, the general health ; secondly, the local conditions present, such as rhinitis and various causes of mechanical obstruction in the nose ; and, thirdly, we often have to deal with mischief set up in the middle ear.

In cases requiring operation we should not delay removal in order that the general health may be improved first, for the operation will in itself largely remove the factors which have resulted in impaired vitality, and general tonic measures will then be much more effectual.

Anæsthetic.—For the removal of growths, in children at any rate, a general anæsthetic is desirable. The choice of the anæsthetic for these operations is at present an open question. In the removal of adenoids alone, the author prefers nitrous-oxide anæsthesia for children who can be persuaded to take it properly. If the tonsils also require removal, one nitrous-oxide anæsthesia is often too short, especially if the fossæ of Rosenmüller should require clearing. Nevertheless, if a previous satisfactory rhinoscopic inspection has made it certain that there is only a central mass in the pharynx, a good administrator will obtain a sufficiently prolonged anæsthesia to permit the post-nasal growth and both tonsils to be removed. Every detail of the operative procedure must be arranged beforehand, as it must necessarily be very rapidly and skilfully executed.



FIG. 148.

Bark's Gag in position. By a little adjustment it can be placed so as not to interfere with the mask for nitrous oxide gas.

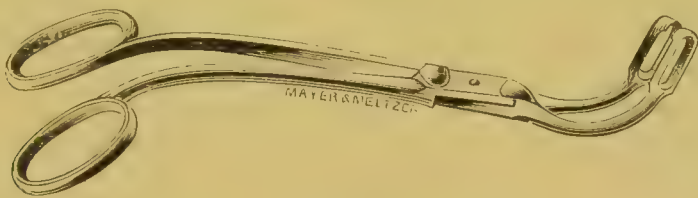


FIG. 149.

St. Clair Thomson's Post-Nasal Forceps.

If for any reason, and this often is the case, a longer anæsthesia is required, chloroform is generally preferable, given till anæsthesia is just complete, care being taken to avoid pushing it to the abolition of the laryngeal reflexes. Bromide of ethyl is very largely employed in America, and on the Continent; it is said, by those who have had very large experience of it, to be safe, rapid, pleasant to take, and not to cause vomiting. Others again prefer ether regularly for adenoid operations, as

there are no special untoward effects from it either during or after operation; it is safer, and therefore to be preferred for certain patients.

The patient should be in the *recumbent position*, with the head hanging well down over the end of the table, so that blood may escape by the nose and not enter the larynx. In older patients the use of cocaine does away with the necessity for a general anæsthetic.

Some operators usually remove the growths with cutting forceps, but it is generally better to use a curette. Gottstein's curette, as modified by Delstanche, is the most useful for removing the large masses in the vault and posterior wall, and has the advantage of being a very safe instrument. It is passed well up behind the soft palate, gently but firmly pressed against the vault and posterior wall, and, as it is drawn down, is made to include and cut through the hypertrophied tonsil.



FIGS. 150 and 151.
Gottstein's and Delstanche's Curettes.



FIGS. 152 and 153.
Hartmann's and Bronner's
Lateral Curettes.

Bronner's curette may be used with advantage for removing adenoids in the fossæ of Rosenmüller, it being safer and less likely to injure the Eustachian orifices than the larger lateral curettes like Hartmann's, which, however, is most useful for removing growths on the pharyngeal vault. After using the curette, the finger should be introduced to ascertain whether any portions of growth still remain for removal, and that the operation is complete and satisfactory. For tough growths it is sometimes necessary to use forceps, and of the many forms of this instrument StClair Thomson's or Dundas Grant's are the ones I prefer.

When both tonsils and adenoid growths have to be removed, first operate on the pharyngeal growths and then the tonsils.

unless the latter are so very large that they interfere with the passage of instruments into the rhino-pharynx, in which case the order of removal should be reversed.

Dangers of the Operation.—Apart from the dangers due to lack of skill or to operating on a struggling child—*e.g.*, injury to the soft palate, the posterior border of the septum and to the Eustachian tubes—there is always the possibility that with the free hæmorrhage a clot of blood may be drawn into the larynx and cause asphyxia. Fortunately I have never had any misadventure in any case, either during or after the operation, but accidents have occurred to the most skilful, and one ought always to have everything ready at hand to do tracheotomy should it become necessary. The chance of blood getting into the larynx is very slight if the head is kept well down, and the anæsthesia is never pushed deep enough to abolish the laryngeal cough reflex.

Violent hæmorrhage may occur from an abnormally-situated internal carotid artery or posterior pharyngeal artery at the time of operation. Secondary hæmorrhage may come on after the operation, and if it trickles down the pharynx and is swallowed, large quantities of blood may be lost without any suspicion of what is occurring. Death has resulted in two instances from profuse secondary hæmorrhage in "bleeders." It may be necessary to plug the post-nasal cavity, and in the more severe and uncontrollable hæmorrhages ligature of the common carotid may be necessary. At least one case of sudden death at the commencement of operation has occurred, the pulse and respiration stopping synchronously without warning (attributed to chloroform neuro-paralysis). In another instance a fatal result was due to convulsions coming on a few hours after the operation was completed; in another, fatal secondary hæmorrhage occurred on the eighth day after operation.

It is well not to minimise the dangers of the operation too much, for though with skill and care the risks are infinitesimal when compared with the enormous benefits accruing from the operation in patients for whom it is really required, many fatal accidents have been recorded. In 1896 Holloway tabulated eleven deaths under chloroform in operations on the tonsils and post-nasal adenoids reported in England between May, 1892, and April 1895, and since then Hinkel has collected nine others,

making twenty in all—six for adenoids alone, three for adenoids and enlarged faucial tonsils, and two for enlarged faucial tonsils alone. In four death occurred before the operation was begun; in three, from a few minutes to an hour after the operation was completed. Doubtless many of the deaths were due to the chloroform, and Hinkel adduces evidence which tends to show that there are some special risks with chloroform anæsthesia in the subjects of lymphoid hypertrophies. For these reasons some anæsthetic more free from risk is desirable when it answers the purpose.

After-treatment chiefly consists in guarding the patient against the risk of catching cold, and thus setting up otitis media, etc., my usual rule being for the patient to remain in bed for one day, in the same room for two days more, and not go out of the house for a further two days. For the first few hours, sucking ice and sipping iced milk lessens any pain or discomfort, and decreases the inflammatory re-action. The food should be light, cold, and soft—such as milk, junket, beaten eggs, custard, and jelly—for a day or two until soreness on swallowing has disappeared.

It is generally undesirable to use any spray or local application for fear of setting up otitis media, and the patient should not be allowed to blow his nose vigorously until several days have elapsed—firstly, because the effort may cause hæmorrhage within a few hours after operation; and, secondly, because there is a risk of driving purulent *débris* into the Eustachian tubes. For similar reasons, in cases where deafness with retracted drums require further treatment, no inflation of the tubes should be commenced until the pharyngeal wound has healed, unless, of course, purulent middle ear disease and perforated drums already exist.

Failure to obtain successful results from the operation may be due to several causes, viz. :—

Firstly, The growths may not be completely removed, enough being left behind to maintain the catarrhal condition and cause rapid recurrence. Incomplete extirpation may be due to an abnormal projection of the anterior tubercle of the atlas, rendering access to the pharyngeal vault and posterior wall above it very difficult unless forceps with a backward projection of the cutting blades are used, such as Quinlan's. Similarly, an excessively developed pharyngeal tubercle or an exaggerated rostrum

of the sphenoid may project from the vault and interfere with the movements of the instrument. Sometimes these rare bony (or cartilaginous) projections come away in the course of operation. Comparatively small lymphoid masses in the fossæ of Rosenmüller or bridges between the Eustachian tubes and the pharyngeal wall may readily be left behind by a careless operator; then, though all obstruction to respiration may be removed, the ear complications will probably persist.

Secondly, Intra-nasal stenosis may co-exist, untouched by removal of post-nasal obstruction. Some chronic rhinitis is generally present in the subjects of adenoids, but usually subsides spontaneously after the cause, viz., the adenoids, is removed. A more common cause of failure, and one to which Harke has directed attention, is the falling in of the alæ nasi from long disuse of the dilator muscles. The anterior nares are small and ill-developed, and during inspiration the alæ are drawn in and act like a valve. The patient should be trained, if possible, to dilate the nares on inspiration, but artificial dilators are useless, for they cannot be worn always. The intra-nasal stenosis may be due to co-existing masses of adenoid tissue on the posterior part of the septum nasi; to hypertrophy of the inferior turbinals; or to nasal polypi, deviated septum, etc., conditions requiring appropriate treatment, described elsewhere.

Thirdly, Buccal respiration may persist simply from habit, the patient may be mentally defective, or deafness and other complications may be pronounced and incurable. In others, the narrowing of the V-shaped palate renders the relation of the upper to the lower jaw abnormal; or the protruding teeth with short lip and weak orbicularis oris may make it difficult for the child to keep the mouth closed, and thus a vacant expression may persist for a long period.

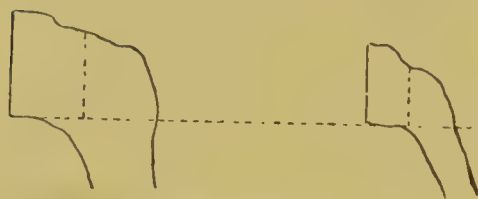
Lastly, Recurrence takes place in some few cases, despite complete extirpation and the absence of any removable sources of failure.

CONGENITAL, NASAL AND RHINO-PHARYNGEAL STENOSIS.

Etiology and Pathology.—Congenital stenosis of the nasal passages may be referred to here in connection with post-nasal adenoids because the facial aspect and other symptoms of these

two distinct affections have in many respects a close resemblance, and it is necessary to differentiate them.

By artificially occluding one nostril in young puppies Zeim has shown that the absence of nasal respiration may result



FIGS. 154 and 155.

Fig. 154 is an antero-posterior section of the healthy rhino-pharyngeal space of a child of ten; it is the mean of seven casts. *Fig. 155* represents the same space of an older child, one aged eleven and a half years, but affected with congenital stenosis. ESCAT.

in defective development of the bones of the corresponding side of the face, but the nasal passages in the human species are sometimes found to be occluded on one side by congenital web formations anteriorly, or by bony stenosis of the choanæ, without any external defect. Yet

if the stenosis is bilateral the facial aspect acquires the defects commonly associated with the post-nasal adenoids. Escat has further drawn attention to cases in which the so-called adenoid facies was associated with congenital stenosis of the rhino-pharynx, without adenoids being present. The patients are generally weak-minded, and hence a careless observer might erroneously attribute the mental defects to the aprosexia of adenoids, whereas careful examination would exclude their existence and reveal the true nature of the defect.

RHINO-PHARYNGEAL CATARRH.

POST-NASAL CATARRH.

Etiology and Pathology. — *Acute rhino-pharyngeal catarrh* usually accompanies attacks of acute rhinitis and pharyngitis from whatever cause arising, but generally disappears with the subsidence of the acute cause, *e.g.*, cold in the head, measles, scarlet fever, etc.

Chronic rhino-pharyngeal catarrh may follow on repeated acute or sub-acute attacks, or may be due to any of the numerous causes of chronic pharyngitis (see p. 81). To these causes must be added neglected adenoids of childhood. The essential pathological factor in true post-nasal catarrh of adults is often disease of the pharyngeal tonsil, and in a considerable proportion of cases it will be found, on rhinoscopic examination, that the pharyngeal tonsil has not undergone the normal involution that commences at the time of puberty, and that adenoid growths

dating from childhood, though partially atrophied, still exist. In many such cases the remains of the pouch of Rathke, the *recessus medius*, is obvious, and this appearance led Tornwaldt to describe the condition as a separate disease.

But it is important to recognise that the so-called catarrh may be due to syphilitic, lupous or tuberculous disease in the rhino-pharynx.

Moreover, post-nasal secretion may come from the nasal passages, or from the sphenoidal sinus, simply passing down into the rhino-pharyngeal space and not arising there. I

believe a large proportion of patients said to be suffering from post-nasal catarrh come under this heading; in yet other cases collections of inspissated secretion are due to atrophic rhinitis and pharyngitis.

Post-nasal catarrh has erroneously been attributed to nasal stenosis and what is termed "negative pressure," resulting from nasal obstruction, but such causes are more or less hypothetical.

The *Symptoms* are similar to those of chronic pharyngitis, together with excessive secretion from the post-nasal space. Tinnitus aurium, deafness, and other evidence of implication of the Eustachian tubes in the catarrhal process are commonly present.

Treatment consists in overcoming any underlying systemic causes, *e.g.*, dyspepsia, constipation, portal congestion, etc. Sea-bathing, a change of air, the administration of tonics, and regulation of the diet are generally called for. Locally, a post-nasal spray or douche with some mild alkaline solution, *e.g.*, Dobell's solution diluted with four parts of warm water, or one compound eucalyptia soloid (B.W.) to the pint of water may be used with advantage once or twice daily. If considerable hypertrophy of the pharyngeal tonsil is present, it should be removed; but it is very important not to attribute the symptoms to any slight departure from the ideal conformation that may be discovered.

The treatment, in fact, which is most beneficial is the same as in chronic pharyngitis.



FIG. 156.

Rhinoscopic image showing a furrowed rhino-pharyngeal tonsil with a "recessus medius," (Rathke's pouch).

CHAPTER XV.

NEOPLASMS OF THE NOSE AND RHINO-PHARYNX.

MUCOUS POLYPUS, AND BENIGN NEOPLASMS—MALIGNANT NEOPLASMS.

MUCOUS POLYPUS.

THE only common form of benign neoplasm of the nasal passages is the mucous polypus. Zuckerkandl found polypi in as many as one out of every eight or nine autopsies in which the nasal passages were carefully examined, from which it must be inferred that they exist in a great many people without producing definite symptoms.

Etiology and Pathology.—The pathogenesis of these polypi is very obscure. They are more frequent in men than women, and though rare before puberty, they are met with even as early as the seventh or eighth year. Morell Mackenzie refuted the suggestion that they are the result of chronic catarrhal conditions, pointing out that polypus is rare before the age of sixteen, while

chronic nasal catarrh is especially common in young children.

Nasal polypi may be primary, or they may be secondary to disease of the accessory cavities. Doubtless there is a causal connection between suppuration in the nasal accessory sinuses and mucous polypi. The ethmoid bone is usually diseased at the seat of the polypi; indeed, Woakes believes that polypus is only a symptom of bone disease, which he terms "necrosing ethmoiditis." But there is seldom any clinical or pathological evidence of *necrosed* bone; what we find is an osteitis, rarefying and formative, as shown in the drawing (*Fig. 157*) of a

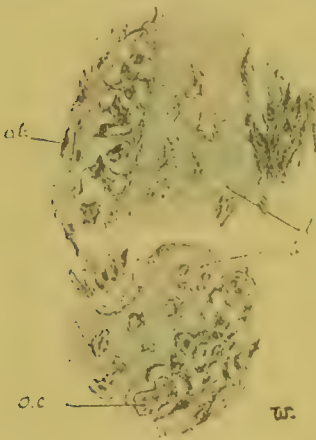


FIG. 157.

Section from the base of a mucous polypus, showing a bony spicule, *b*; with osteoblasts, *ab*; and osteoclasts, *oc*.

section from the base of a mucous polypus. Zuckerkandl in reference to this question states that he has not observed necrosis of the

bone in a single case, but that he has seen the bony part which exists in the base of some polypi become elongated and softened. The layers of the periosteum undergo inflammatory changes which result in this *osteitis* and in hyperplasia of the fibrous connective tissue, by œdematous infiltration of which the ordinary polypus is formed. Thus we may often detect rough or bare bone, or a sense of softened carious bone conveyed by the contact of a probe with the bony trabeculae at the base of the polypi. It seems probable that obstruction arises in the lymphatic vessels owing to the invasion of micro-organisms (although I have not succeeded as yet in obtaining histological proof of such a view, and thus inflammatory and degenerative changes are induced, which originate the sodden, water-logged mucous membrane so often associated with mucous polypi. Some alteration in the vascular supply, with consequent exudation and œdema is believed by Hopmann to be the prime factor in their causation.

The (so-called myxoma) mucous polypus is composed of fine meshes of areolar tissue filled with fluid containing serum-albumen and mucin, and covered with the ciliated epithelium of the mucous membrane when small, though this normal ciliated epithelium is generally lost and replaced by stratified epithelium as the polypus becomes larger. It contains a variable amount of glandular tissue, generally only a few mucous glands being present, yet only very rarely is the glandular element so preponderant as to constitute a fibro-adenoma.

From cystic degeneration of the mucous glands we get one form of cystic polyp, but in other polypi, in which the glandular element is almost or completely absent, simple liquefaction of the contents may give rise to one large cyst. Thus we may differentiate the varieties of mucous polypi by the terms:—

Fibroma-œdematosum	Fibro-adenoma-œdematosum
Cysto-fibroma-œdematosum	Cysto-fibro-adenoma-œdematosum

The exposed portion of a polypus from frequent irritation will sometimes develop a papillomatous structure, but papilliform œdematous polypi are met with in association with ordinary œdematous polypi. Jon. Wright in a series of specimens has observed histologically all stages of an apparent transition from the ordinary smooth form to the papillary form, in which the surface and the glandular epithelium alike proliferate.

Though from cases coming under treatment we find that mucous polypi nearly always arise from the middle turbinal, or from the margin of the *hiatus semilunaris*, Zuckerkandl found from *post-mortem* examinations that they are very frequently situated in the superior meatus, or on the superior turbinal.

These mucous polyps fall into two distinct pathological groups:—

(a) The origin of the polypus is most frequently associated with, if not actually due to, inflammation of the mucous membrane, resulting in hyperplasia of its tissues, including the periosteum. The infiltrated area becomes œdematous and vascular, and a fibrous stroma is formed from the connective tissue cells. In other cases associated with suppuration, granulation tissue is formed and becomes œdematous and vascular, the ciliated epithelium creeping over the granulation polypus from its base, as described by Macdonald, just as granulation polypi in the ear become covered with the squamous epithelium of the tympanum.

(b) But another group, and by no means inconsiderable proportion of nasal polypi, are not associated with inflammatory changes, but with recurrent vaso-motor phenomena of nervous origin. Thus localised œdematous infiltration, often amounting to a polypus not rarely follows or arises in the course of attacks of asthma, and with frequently repeated asthmatic paroxysms the polypoid mucosa tends to persist, and once the polypus is originated it tends to increase in size, and perhaps its dependent position by mechanical action co-operates with the vascular changes in determining its subsequent development.

Symptoms.—The most prominent symptom is stuffiness or obstruction in the nose, varying, of course, with the size of the polypi. The patient can often feel something which “flops” to and fro on inspiration and expiration, and may be distinctly conscious of some loose body in the nasal passages.

The nasal secretions are in excess, and the constant sniffing and running at the nose are extremely annoying. If nasal respiration is much obstructed, mouth breathing with its attendant evils is present, the voice becomes nasal, muffled and without resonance, and the sense of smell is lost. As the growth increases in size it may press upon and obstruct the orifices of the accessory sinuses (with consequent retention of the

secretions of the antrum, frontal sinus, etc.)—or of the nasal duct, giving rise to lachrymation and epiphora. Mucous polypi, if large, often cause deviation of the septum, but while the nose may appear broadened from some œdematous infiltration, external bony displacement is hardly ever seen.

Patients generally find the symptoms aggravated in damp weather, partly owing to the hygroscopic properties of the polypi, and partly to the effect of damp on the rhinitis which almost invariably co-exists. Catarrhal deafness may supervene from associated naso-pharyngeal catarrh, or from direct pressure on the Eustachian tubes.

True nasal neuroses are not uncommonly set up. Thus Hack enumerates nightmare, persistent cough, hemicrania, and epilepsy, as especially liable to occur when the anterior inferior portion of the lower turbinal is pressed on, while, as in Volto-*lini's* classical case, asthma is sometimes due to the presence of a polypus. (See "Nasal Neuroses.")

Objective examination seldom leaves any room for doubt as to the diagnosis. If the polypus has attained any size, and is situated anteriorly, it will be seen as a characteristic semi-translucent, smooth, greyish, gelatinous body, occupying the middle meatus, or reaching down to the inferior meatus, or even presenting at the anterior nasal orifice. Unless very large it is generally possible to determine its seat of origin, which, as stated above, is generally beneath or from the free border of the middle turbinal. Less frequently its attachment is higher up, but never does it originate from the inferior turbinal, and hardly ever from the septum.

When the polypus is large only one may be seen, but they are generally multiple, and, more often than not, bilateral. Mucous polypi are sometimes very large. Thus *Zaufal* reports the removal of one more than 6 inches in length, and weighing over $3\frac{1}{2}$ ounces; and *Delie* had a patient from whom he removed a mass of polypi weighing 103 grammes—63 grammes from the right nostril, and 40 from the left. In many cases no large polypus is present, but there are numerous small polypi the size of currants, or smaller, beneath or attached to the free edge of the turbinal. Sometimes the free border of the middle turbinal has undergone polypoid degeneration when no definite pedunculated polypus is present.

When a polypus is deeply seated, it may often be made to come to the front by the patient blowing the nose vigorously. But when thus growing far back, or when the polypus has



FIG. 158.

Posterior rhinoscopic view, showing moriform hypertrophy of the inferior turbinals, partly concealed on the patient's right side by a smooth mucous polypus. Hypertrophic tissue is likewise seen on the vomer.

extended backwards towards the posterior nares, the diagnosis can only be made by a posterior rhinoscopic examination, which shows the polypus protruding from the choanæ into the rhino-pharynx, or even completely filling it, so that it hangs down and appears below the soft palate.

There should be no difficulty in distinguishing a polypus in this situation from (1, hypertrophy of the inferior turbinal; 2, symmetrical adenoid growth on the septum; 3, post-nasal adenoids; but it is not easy to make a diagnosis by inspection alone from those rare cases of fibrous polypi growing from the nasal cavities towards the rhino-pharynx.

Large polypi, which have undergone compression by surrounding structures, tend to lose their usual cedematous appearance, and become more solid and fibrous on the surface, and, if coming well into the anterior nares, may be red and lobulated, and simulate papillomata in appearance.

Examination of these polypi with a probe will always show them to be soft and freely moveable, and that they are not connected with the septum or inferior turbinal, while they are readily penetrated by a sharp needle.

Diagnosis.—Mucous polypi must be differentiated from fibrous polypi, cancer, and sarcoma, all of which are painful, bleed freely when probed, are firm in texture, and, if large, produce bony displacement. Chronic abscess of the septum and bloody tumour, which results from blows, are generally bilateral, and are situated on the septum. Papilloma, hypertrophy of the lower turbinated body and foreign bodies, will hardly give rise to confusion on careful examination, while cartilaginous and osseous tumours are hard and present other distinguishing characteristics. Primary carcinoma of the nasal fossæ is exceedingly rare.

It is most important that the co-existence of accessory sinus disease should not be overlooked, and careful investigation should be made in reference to this question in every case of polypus.

Treatment consists (1,) in removal of the polypi, and (2,) treatment of the abnormal conditions which have caused their growth.

The only methods of removal which merit discussion are (*a*,) avulsion, and (*b*,) snaring.

Avulsion, by seizing the polypi with suitable forceps, has been extensively condemned by many specialists, who advocate the use of the snare as the only justifiable means. To blindly introduce forceps and tear away whatever happens to have come within their grasp is indeed a disastrous procedure, but when the position of the polypus has been made out, and forceps are used with skill and care, they are a very efficient means of getting rid of the growths. There are two conditions in which they must be resorted to, viz., when it is impossible to introduce the loop of a snare owing to the size or situation of the polypus, or when a number

of small sessile

polypi occupy the middle meatus and cannot be snared. These small polypi should be removed one by one by small forceps. For larger polypi, Mackenzie's punch forceps or broad-bladed, serrated forceps with a catch, are sometimes more suitable; but even large polypi may be rapidly and easily removed by small forceps which can be insinuated between the body of the growth and the nasal wall. It is essential that the polypus should be seized as near its attachment as possible, and the growth is detached by traction and twisting, so as to avoid the risk of dragging away large portions of the bony structures. In addition to the danger of tearing away large pieces of the turbinated bones, the disadvantages of the forceps are the pain that their use involves and the hæmorrhage. Bleeding generally ceases in the course of a few minutes, and may be checked by spraying with cold water. If hæmorrhage continues to be free, it may be necessary to plug the nasal passages. (See "Epistaxis.")

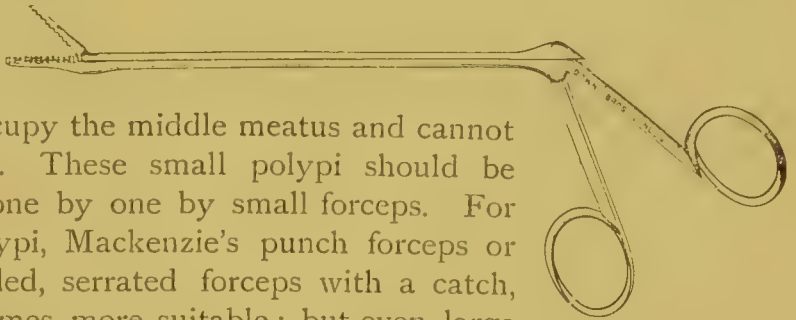


FIG. 150.

Alligator Forceps for removal of small sessile polypi.

Of course, before polypi are removed, cocaine (10 or 20 per cent.) should be used. This should be applied, as far as possible, to the root of the polypus and to the mucous membrane of the nasal fossa, and not simply sprayed on to the polypus itself, which is practically devoid of sensation. For this purpose a spray with a fine nozzle that can be introduced between the polypus and the inner and outer wall of the nares should be used. In young children it is sometimes necessary to use a general anæsthetic. The posterior nares should then be plugged, if possible, to prevent the escape of blood into the rhino-pharynx with the necessity of constant mopping.

Snaring is the method of removal that may often be adopted with advantage, as it is often less painful and involves less hæmorrhage.

A generally useful form of snare is the one devised by Lack,

as the loop can be tightened quickly or slowly as desired, and can always be drawn out readily and re-introduced without the trouble of changing the wire.



Fig. 160.
Lambert Lack's
Nasal Polypus
Snare.

A No. 5 to 10 steel piano-wire is generally used for the cold snare. After the application of cocaine, the nostrils being dilated by a nasal speculum, the loop of the snare, which must, of course, be large enough to pass over the whole

polypus, should be introduced vertically, the upper part of the loop being insinuated between the outer wall of the nasal passage and the growth. Then, turning the snare to the horizontal, it should be passed up to the root of the polypus by gentle movements, and gradually tightened as the narrow pedicle is reached. The loop having thus been placed round the pedicle as near the base as possible, it should be tightened till the pedicle has been cut through, when the growth can be removed.

A polyp which is situated far back or has passed into the rhino-pharynx, often proves most difficult to snare. If it can be brought forward by the patient blowing his nose, it may be seized with a hook or small forceps, and held while the snare is passed over the hook or forceps, and thus made to encircle the

pedicle. It is sometimes possible to pass the snare along the floor of the nasal fossa to the rhino-pharynx, and then, with the aid of the forefinger in the mouth, to get the snare round the dependent growth. In most cases the forceps passed well back can be made to seize the pedicle, and the polypus be got rid of in this way with less discomfort to the patient.

The galvano-cautery snare may be used instead of the cold wire snare, the loop being placed in position in the same manner as for the ordinary snare, and the current passed through the wire as it is being tightened. It is said to be followed by less hæmorrhage, and to have a greater tendency to prevent recurrence. But the cold wire snare can be employed without involving much bleeding, provided it is used slowly and skilfully, while it is always necessary to cauterise the stump, even if the cautery snare has been used. Moreover, the cautery snare is sometimes followed by considerable inflammatory reaction from the unnecessarily extensive scalding of the contiguous mucous membrane as it is burning through the pedicle. For these reasons it has largely fallen into disuse, except for those polypi with thick, firm pedicles that the cold snare will not cut through. When the passages are very narrow it is occasionally necessary to remove the anterior inferior portion of the middle turbinate by scissors or snare, as a preliminary measure.

To prevent the recurrence of the growth it is usually advisable within a day or two to deeply cauterise the pedicle with the galvano-cautery, or to apply chromic acid by means of a probe. The mucous membrane around the pedicle is generally in a more or less polypoid condition, and should be cauterised as well. Indeed, the small, currant-like polypi which sometimes surround the opening of the *hiatus* are often best treated by the application of the cautery alone. The patient should then be directed to use an oil atomiser with some antiseptic in solution, *e.g.*, eucalyptol or terebene, grs. x to xx to the ounce of liquid vaseline. It is essential that the patient be kept under observation for some time, as even when all growths have apparently been removed at the time of the operation, others will often become obvious in the course of a day or two, after the larger mass, which has compressed or displaced them, has been removed.

Of course when the nasal polypi are secondary to, or are

associated with suppurative inflammation in any of the nasal accessory sinuses, it is essential that the implicated sinus should be treated. In many cases the ethmoidal cells are thus diseased, and then it is necessary to open, freely curette, or partially extirpate these cells in order to obtain a lasting and radical cure.

The other benign neoplasms occasionally met with are :—

FIBROMA, an exceedingly rare growth in the nose, but fairly common in the rhino-pharynx. Of twelve intra-nasal cases occurring in the nose, collected by Casselberry, in one (his own), the fibroma was attached to the ethmoid, the others to the roof, cribriform plate, upper turbinal, and septum. They are firm,

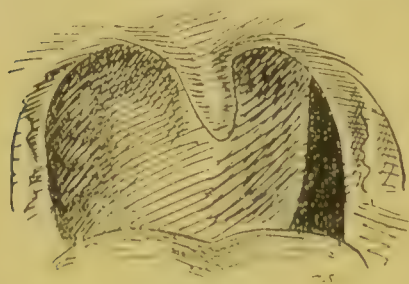


FIG. 161.

Large fibrom growing from the rhino-pharynx.

compact, painful, bottle-shaped growths, covered with smooth light pink or red mucous membrane, and are generally attached to the periosteum of the basilar process of the occipital bone of the body of the sphenoid, of the vomer, or to the septum. As they bleed readily, in addition to the usual symptoms of nasal

polypus they are attended with epistaxis. Rhino-pharyngeal fibroma very rarely occurs before puberty or after the twenty-sixth year in fact this disease is almost confined to males between the ages of fifteen and twenty-five. Lincoln, of New York, collected fifty-eight cases, all being in males under twenty-five. When removed in early life they tend to recur up to the age of twenty-two, but they may atrophy spontaneously in early adult life after attaining large dimensions. They may be removed by the galvano-cautery or cold wire snare, or by electrolysis if small. Hæmorrhage is often very copious, and when large it is usual to have recourse to splitting the soft palate, and, if necessary, partial removal of the hard to give room for the snare. The reader is referred to works on general surgery for the removal of these growths in the pharynx.

PAPILLOMATA are very rare in the nose. They always grow from the lower turbinal or the septum, vary in size from a pea to a filbert, and are often multiple. They are pink or red, soft,

lobulated, raspberry-like, sessile or pedunculated growths which are moveable and bleed readily. They are papillomatous in structure, having a thickened epithelial layer of squamous epithelial cells, the central interstitial tissue being composed of connective tissue and a few blood-vessels. Sometimes they are very vascular, and have then been described as angiomata. It is necessary to distinguish carefully true papilloma from Hopmann's polyp, the papillary œdematous hypertrophy of the nasal mucous membrane not infrequently associated with ordinary mucous polypi, to which reference has already been made. I have only met with two cases; one was single, the other multiple and bilateral, and in the latter case there was (*Fig. 162*) a smooth deep-red vascular tumour the size of half a spanish nut occupying the septum opposite the middle turbinal, similar to those reported by Verneuil, Schadowald, and Flateau. Papillomata should be removed by the snare, or by the application of chromic acid. Fletcher-Ingals has found the application of *thuja occidentalis* reduces the growths and tends to prevent their recurrence, which is very liable to arise.



FIG. 162.
Papilloma of
Septum.

EXOSTOSES are uncommon, and rarely attain any size. A history of syphilis, congenital or acquired in many of the cases, points to the probable cause of the affection. They grow from the floor, or from the lower part of the septum, when they simulate deviated septum. They are hard and painless, but may cause obstruction.

OSTEOMATA are sometimes found. There are two varieties, the hard ivory and the soft or cancellous, the former being the most frequent. They grow from the periosteum of the septum or turbinated bones, and are often slightly moveable, being attached by a short firm pedicle. The surface is covered with red uneven mucous membrane, and by growth in various directions they may cause displacement of bony structures. They may often be removed by forceps or snare, but sometimes come away spontaneously from atrophy of the pedicle. In one of my patients the only symptom was gradually-increasing obstruction for twelve months. The left nasal passage was completely occluded, but only the small extremity could be seen by anterior rhinoscopy. Nevertheless, when removed by my colleague,

Carwardine, it proved to be a large ivory osteoma, four inches long, attached to the septum close to the nasal floor.

ECCHONDROSES are very rare, occurring generally in young males on the septum, and cause obstruction, catarrhal symptoms, and, if large enough, external deformity. If not too large they should be removed by the cold or the galvano-caustic snare.

CYSTS.—Besides the various forms of cystic polypi already referred to, we may rarely meet with cystic growths from the turbinal bodies, and air-containing cavities in the middle turbinal bone may arise from hypertrophy with incurving and attachment of the free border of the turbinal due to osteophytic periostitis. Brown Kelly has collected twelve recorded cases of cysts on the floor of the nose. The patients were all females between the ages of nineteen and fifty-eight. A swelling is first observed by the patient beneath the ala, and then having attained a certain size, remains stationary, or rapidly enlarges and becomes painful, varying in size from an almond to a walnut, and when large causing external deformity. Kelly considers them to be retention cysts. Hydatids and dermoid cysts have likewise been met with in the nasal passages.

MALIGNANT NEOPLASMS.

Sarcoma is rarely primary in the nose, while *carcinoma* is extremely rare. A sarcoma sometimes takes the form of a mucous polypus, which it may so closely simulate as only to be differentiated by microscopical examination. I have known a patient, with mucous polypi recurring for some years, develop nasal sarcomatous polypi, an instance of the occasional sarcomatous degeneration of fibromata.

Symptoms.—Primary malignant growths generally develop on the septum. Sarcoma occurs as a single, sessile, soft, lupus-like deposit, with smooth or rugose surface of pink or brown colour, rapid in growth, and very vascular. Cancer, epithelial or encephaloid, commences as a small wart-like growth of dark red or purple colour, and generally ulcerates early.

A sarcoma affords more chance of radical extirpation than a carcinoma, and a fibro-sarcoma appears to be more favourable than a pure sarcoma. Sarcoma of the nose generally occurs in elderly people, but may arise at any age. Frequent and severe epistaxis associated with a sessile septal polyp is very suggestive of sarcoma in patients over forty.

Malignant growths are attended with foetid discharge, pain, epistaxis, and grow rapidly. In the early stages, with small defined growth, radical removal would offer some chance of extirpating the growth, but the prognosis depends largely on the degree of malignancy and the seat of the growth.

TABLE FOR DIFFERENTIAL DIAGNOSIS.

<p><i>Chronic Hypertrophic Rhinitis.</i></p> <p><i>Age:—</i> Adults, especially males</p> <p><i>Symptoms:—</i> Nasal catarrh and obstruction, discharge muco-purulent, and naso-pharyngitis. No pain. Hypertrophy of the inferior and middle turbinals, especially posteriorly, where they form spawn-like masses in the choanæ. No ulcer, no hæmorrhage.</p>	<p><i>Atrophic Rhinitis with Ozena.</i></p> <p><i>Age:—</i> Puberty and young adults, especially females.</p> <p><i>Symptoms:—</i> Loss of smell and intermittent discharge of intensely foetid mucus, and nasal obstruction by dry crusts; both smell and obstruction removed by cleansing. Olfactory fissure wide, bridge of the nose often depressed. Atrophy of turbinal bodies. No ulcer, no hæmorrhage.</p> <p>No destruction of bone.</p>	<p><i>Syphilis.</i></p> <p><i>Secondary:—</i> Occurs with manifestations of syphilis in other parts.</p> <p><i>Tertiary:—</i> Gumma, nasal obstruction persistent, no pain, little discharge. Is a large smooth, red, hard, and elastic tumour. It soon ulcerates with muco-purulent discharge.</p> <p>Ulcer often longitudinal, deep, edges thickened, with pale granulations, which readily bleed. Margins slightly raised, surrounded by areola; floor covered with pus. Not tender to touch. Does not bleed easily. Destruction of bone as well as cartilages. Syphilitic neoplasms are very amenable to treatment by internal remedies.</p>
<p><i>Tubercle.</i></p> <p>Generally associated with tubercle elsewhere.</p> <p><i>Symptoms:—</i> Nasal obstruction absent or slight. No pain. Neoplasms. Ulcers small, round or ovoid, or irregular outline, margins not raised, surrounded by pale mucous membrane, covered with greyish-white opalescent muco-pus, surrounded by pale mucous membrane. Not tender to touch, and not great tendency to bleed.</p>	<p><i>Lupus.</i></p> <p>Generally associated with lupus of skin, in the young chiefly.</p> <p><i>Symptoms:—</i> Nasal obstruction. No pain, or only slight. Clusters of small, red, firm, elastic growths of slow progress. Ulcers—margin raised, covered with crusts, tends to cicatrize, surrounded by normal mucous membrane. Bleed easily on removing crusts, and are painful when touched. Carries of soft structures only.</p>	<p><i>Benign Growths.</i></p> <p>Polypus freely moveable, gelatinous, translucent, pedunculated growths from the middle or superior turbinal bodies or meatuses. Generally multiple.</p> <p><i>Symptoms:—</i> Nasal obstruction or discharge. No pain.</p> <p><i>Fibroid:—</i> A single, firm polypus or sessile growth from septum or inferior turbinal. Painful.</p> <p><i>Papilloma:—</i> A cherry red or pink moriform growth on the inferior or turbinal septum, often multiple. Painless nasal obstruction the only symptom.</p>
<p><i>Malignant Growths.</i></p> <p><i>Age:—</i> Advanced. Sarcoma at any age.</p> <p><i>Symptoms:—</i> At first nasal obstruction and epistaxis, rather than pain.</p>	<p>Later, intermittent, lancinating pain. Constitutional cachexia. Growths generally unilateral, single, springing from septum, are sessile, red or purple, soft, bleed on touch, not tender. Rapid growth and early ulcer-</p>	<p>ation, with discharge of sanious greenish muco-pus, soon becoming foetid. Microscopical examination important. Ulcer deep, irregular, covered with muco-pus.</p>

CHAPTER XVI.

DISEASES OF THE NASAL SEPTUM.

PERFORATION, DEFLECTIONS—EPISTAXIS—ABSCESS—FRACTURES.

PERFORATION OF THE SEPTUM.

PERFORATION of the septum was found by Zuckerkandl eight times in 150 autopsies, and is, therefore, more frequent than is generally supposed. In many cases it causes no symptoms and is without significance, and certainly it cannot be held that perforation of the cartilaginous septum is a proof of syphilis. The two chief sources of inconvenience are: (1), the tendency for secretion to collect and form crusts on the margins of the hole; and, 2, a whistling sound during respiration if the hole is small and situated far forward; large perforations do not cause "whistling." It may be due to simple ulceration in dry rhinitis, from picking with the finger nail, to idiopathic periostitis, or result from an abscess following a blow. Irritating dust such as arises in chrome works often causes septal perforation. Perforation sometimes occurs in lupus and malignant disease, and then the cause is obvious. But the commonest cause is syphilis.

When only the cartilage is involved symptoms are often absent, but if the perforation involves the bony septum, fœtor is almost invariably present in very marked degree. The bridge of the nose only becomes sunken when the nasal bones are diseased. I have known the whole septum to have completely disappeared without any falling in of the nose at all.

SEPTAL DEFLECTIONS; CRESTS, AND SPURS.

Deviation of the septum in some degree may almost be regarded as its normal condition, for it is only in comparatively few persons that it is absolutely straight. Morell Mackenzie, in an examination of 2,152 skulls in the Royal College of

Surgeons, found a deviated septum in 1,657, or about 75 per cent. In about 40 per cent. of the cases the deviation was to the right, in 30 per cent. to the left, while the remainder were sigmoid or zigzag. Mayo Collier, from an examination of over 1,000 patients, states that in adults not more than 10 per cent. are without some septal irregularity, and that only in young persons under puberty may we expect to find a majority of normal septa. In some cases, however, the deviation causes such marked symptoms, either from simple mechanical obstruction and its consequences, or by setting up nasal neuroses, that the condition calls for treatment.

Etiology and Pathology.—As regards the etiology of these deformities of the septum, there is much difference of opinion. Morgagni and others believe that the deviation is frequently congenital, but Zuckerkandl, in his elaborate investigations, found none under seven years of age. Zuckerkandl and many rhinologists agree in attributing to blows and injuries the chief responsibility for the condition, and my own observations have led to the same conclusion. Mayo Collier urges that the negative pressure or suction exerted during inspiration through the nasal passages is the essential factor in the production of deflections, because whenever one nostril is more stenosed than the other, this suction force is more marked on that side, and the septum in a longer or shorter time yields to the pressure; but he also attributes many cases to blows.

The deflection, in patients presenting symptoms, is generally most marked in the triangular cartilage, especially in its anterior portion. It may be a simple twist of the anterior end to the right or the left, or it may be a sigmoid deflection, obstructing alternately the right and left passages.

As the result of perichondritis from a variety of causes, *e.g.*, fracture of the cartilage or its dislocation from its attachment to the vomer, a cartilaginous ridge may be formed antero-posteriorly or obliquely. A deflection on one side generally has a corresponding depression on the other.

In other cases we find simple kinks, spurs, or rounded prominences projecting into the passage, and approaching or coming into contact with the lower turbinal.

Symptoms.—As a consequence of the partial nasal obstruction from deflected septum, inspiration causes a rarefaction of the air

behind the obstruction : as a consequence over-filling of the vessels, and constant hyperemia with chronic rhinitis and further increase of the nasal stenosis may result.

The chief symptoms are those of nasal obstruction, viz., buccal respiration and its consequences. In not a few cases the unilateral catarrh behind the obstruction causes Eustachian catarrh and deafness of the corresponding ear. Again, the other nasal passage, even if normally patent, is often unequal to moistening the whole of the inspired air, and, therefore, is liable to inspissation. Thus, in a patient recently sent to me by a medical practitioner, the chief complaint was pain and dryness of the left nasal passage, for which he had been recommended to artificially close this side, especially on going to bed. The whole trouble was due to a deviation of the septum occluding the right passage, and, therefore, the condition of the patient was, of course, only aggravated by closing both sides.



FIG. 163.

Septal deflection and well-marked spur, associated with hypertrophic rhinitis, paroxysmal sneezing and asthma.

It may be found that in addition to the symptoms caused by rhinitis and post-nasal catarrh, the patient is very liable to repeated attacks of bronchitis and laryngitis. As a consequence of rhinitis and the extreme irritability of the nasal mucosa set up by a septal spur, patients sometimes suffer from paroxysmal sneezing or other nasal neuroses.



FIG. 164.

Septal deflection with nasal obstruction, due to bony prominence, in association with turbinal enlargement.



FIG. 165.

The same after cocaine has caused shrinking of the turbinates, the bony prominence and the septal deflection remaining unaltered.

Diagnosis.—This should be obvious on examination of the nasal passages. The only conditions that are liable to cause confusion are syphilitic perichondritis, and hypertrophy of the

vascular tissue of the septum. The former is inflammatory, the latter is soft and fluctuating and is reduced by the application of cocaine.

Treatment.—The treatment of these septal deformities is only called for when definite symptoms demand their removal, and very often they are best left alone. But when it does become desirable to rectify the defect, various methods may be con-



FIG. 166.

Curtis's Trephines for septum.

sidered, for no one procedure is applicable to all alike. For this reason it is necessary to dwell at some length on certain of the operations that are in vogue, although it is equally necessary to protest against the altogether undue importance that is sometimes attributed to perfectly harmless spurs and septal deflections. In cases where there is only a narrow ridge, the simplest method of relieving the condition consists in reducing it by means

of the gal-

vano-cautery.

In a great

many patients

the obstruc-

tive symptoms are due to an associated hypertrophy

of the inferior turbinated body, and sometimes the

treatment of the turbinal hypertrophy (see p. 286), is

all that is needed. But if the turbinal body is normal,

it is most undesirable to relieve nasal stenosis by

destroying such an important physiological structure,

and therefore, unless amputation of the anterior extremity is

sufficient to relieve the obstructed passage, it is often necessary

to remove the deflection (the ridge or spur) by mechanical

means. We may then resort to one of the many devices

for rectifying the deformity, such as MacDonald's gouges,

Bosworth's nasal saw, Seiler's burrs, Curtis's trephines, or

Major's knife.

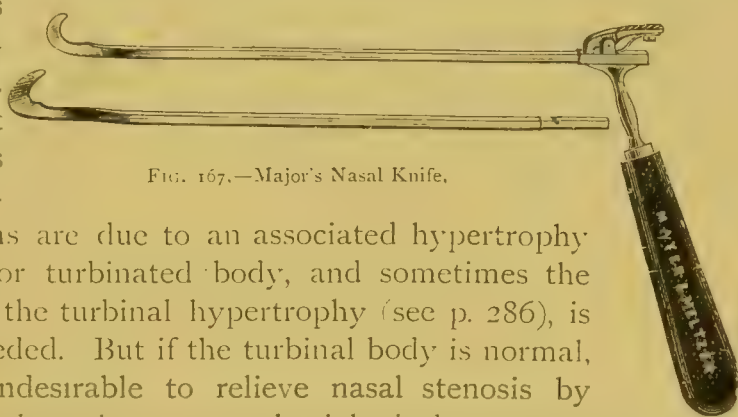


FIG. 167.—Major's Nasal Knife.

MacDonald's method is as follows: "A single linear incision is made over the most prominent point of the neoplasm, well down to the cartilage. With a raspatory the perichondrium, with

its inseparable mucous membrane, is then turned up and down sufficiently to expose the portion to be removed. Next, the superabundant cartilage is separated with a gouge, or saw if it prove to be ossified. Finally, the flaps are allowed to fall together, the wound is dressed with iodoform, and a small tampon of cotton-wool, impregnated with some antiseptic, is inserted so as to exert a gentle pressure upon the flaps, and assist in retaining them in their position. Healing frequently takes place by first intention." (Diseases of the Nose.)

Favourite instruments are Carmalt Jones' spoke-shave, and Major's nasal knife, which are placed in position behind the spur, and then firmly drawn forward, cutting through the obstructing cartilage. These knives have been specially designed for that class of case in which a long crest or spur is found running in a horizontal direction from before backwards along the lower third of the septum narium near the floor of the nasal chamber. There are numerous other instruments, introduced by different rhinologists, but they are mostly modifications of one or the other of the forms already alluded to. Adams' or Hewetson's septum forceps, or Hill's dilator, should only be used when it is desired to correct a bony deformity, or when a deviation causes external deformity, for they can hardly be expected to relieve obstruction so effectually as the methods of removing the redundant or obstructing tissue.



FIG. 178.
Hill's Dilator.

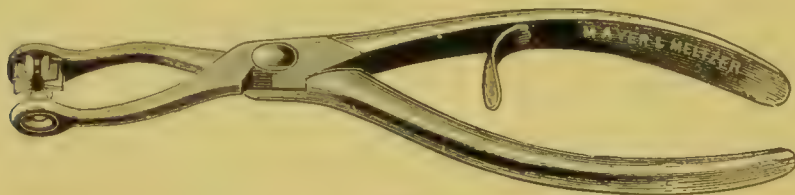


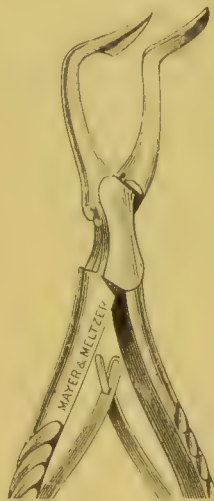
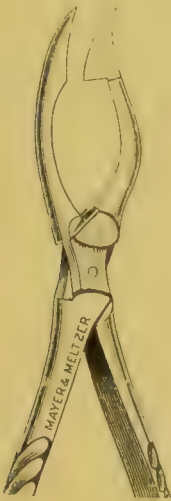
FIG. 109.
Jarvis-Steel Septum Forceps.

Punches, such as Steele's, I should think, are liable to cause sloughing and perforation.

Bryson Delavan first elevates the mucous membrane, and then if the soft tissues are replaced after removing the spur, etc., a perforation is avoided. Escat, of Toulouse, has succeeded in

detaching the mucous membrane of the concave side by injecting sterilised water under it by means of a hypodermic syringe. He then removed the projection by means of a bistoury. De Blois strips the perichondrium and mucous membrane from the spur, saws off the spur, and then fastens down the flap of membrane by means of collodion.

Asch's operation for deviations of the cartilaginous septum is comparatively simple, and has proved very successful not only in his own practice but in the hands of many others of his colleagues, notably Mayer, who reports two hundred collected hospital cases.



FIGS. 170 and 171.

Asch's Horizontal and Vertical Cutting Scissors, for operating on septal deflections.



FIG. 172.

Asch's Blunt Forceps.

The operation is performed under complete general anaesthesia. The nose having been cleansed by some alkaline antiseptic solution, the blunt separator is introduced on the deviated side to break down any adhesions between the septum and outer wall. Then the straight scissors are introduced, the blade in the concavity and the blunt edge over the point of greatest convexity, parallel to the floor of the nose. They are firmly closed, the blade cutting through the cartilage with a snap. The angular scissors are then introduced, this time pointing upwards towards the frontal bone, so that when closed their incision shall be as nearly as possible at right angles to, and crossing the centre of the first incision. The finger is then introduced in the deviated side, and the four segments made by the incisions are forcibly pushed into the concavity, effectually breaking them at their base. The blunt forceps are then introduced, one blade in each nostril, and firmly closed, thus straightening the septum and forcing the broken segments to over-ride each other in the concavity. Sterilised vulcanite hollow

plugs are introduced in the nose, a snugly-fitting one on the stenosed side, and a smaller one in the opposite nostril, so as to cause equal pressure. The operation is then complete, the bleeding being always promptly checked when the pressure is applied. The patient is kept in bed for a day or two, and iced cloths applied externally. Twenty-four hours later the tube in the concave side is removed and not replaced, and after another twenty-four hours the tube on the stenosed side is removed and cleansed, the passage being cleaned with the aid of cocaine if necessary, and the tube is then re-introduced. The tube is removed and cleansed each day, until about a month from the day of operation it is finally withdrawn.



FIG. 173.
Mayer's Hollow Nasal Plug.

Chiari for simple deviations resects the septal cartilage in the form of a triangular flap, and keeps the flap in an over-corrected position by means of iodoform plugs for two or three weeks. A somewhat similar method, devised by Hajek, is suitable for simple deviations of the anterior portion of the cartilage.

Moure, of Bordeaux, strongly advocates electrolysis, and in the discussion on his paper at the Rome congress Rosenfeld supported him in this practice, stating that "cartilage melts away under its action like butter under a hot sun. Only one operation is needed, and this is applicable to bony growths as well, if the bone is not too dense to be penetrated by a needle."

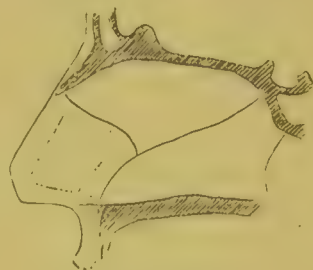


FIG. 174.

Diagram showing the method of Hajek by making incisions along the dotted lines so as to include the deviated portion of cartilage with the mucosa attached, and keeping the then moveable portion in the median line by antiseptic gauze plugs.

The bipolar method is used—two separate, straight steel needles, sterilised, insulated to within half an inch or so of the ends, are inserted one above the other into the spur, parallel and separated by the greatest interval the spur allows, and a current of from 15 to 20 ma. passed for about fifteen minutes. If bubbles of gas appear beneath the mucosa, the current is turned off as a perforation is threatening. It should be reversed for a few minutes before withdrawal of the needles to check hæmorrhage. The most central needle is connected with the positive pole.

As a rule cocaine is the only anæsthetic required for electrolysis, and as hæmorrhage is generally rather free it presents

special advantages in that by its use we lessen the difficulties that such hæmorrhage causes when the operation is done under general anæsthesia.

If forceps are used, or if the portion to be trephined is considerable, a general anæsthetic will be necessary, as the operation is painful. When a considerable surface removal is necessary, it is desirable to reflect the mucous membrane before removing the underlying cartilage, otherwise an extensive cicatrix is formed, and causes dryness of the passage and discomfort to the patient.

EPISTAXIS, HÆMATOMA AND FRACTURES.

HÆMORRHAGE from the nose may be due to (1,) *injuries* from blows, etc.; (2,) *local affections* of the nasal passages, hyperæmia, primary or secondary to cerebral congestion, or from ulceration in malignant disease, or from foreign bodies; (3,) *systemic affections*, such as anæmia, purpura, scurvy, Bright's disease, portal congestion, cirrhosis of the liver, enteric fever, measles, scarlatina, diphtheria, pneumonia, etc.; or (4,) it may be vicarious and occur at the menstrual periods only.

Sources of Epistaxis.—(a,) The hæmorrhagic spot is most frequently at the anterior inferior part of the septum, but when from this source, bleeding is rarely so profuse as to be dangerous; (b,) The bleeding point may be at any part of the nasal fossæ, or there may be general oozing from numerous points, as is usually the case in epistaxis due to purpura, hæmophilia, and toxæmic conditions. If the bleeding is from the anterior part of the passages, the blood escapes from the anterior nares; but when it is more deep in origin it passes into the naso-pharynx, and may be swallowed and subsequently vomited in large quantities, or it may pass into the glottis and be coughed up and thus simulate hæmatemesis or hæmoptysis; (c,) In a third class, to which Brown Kelly has directed attention, the site of the bleeding is from the upper and anterior region of the nasal passage, above the middle turbinal. These cases, he believes, are due to rupture of the anterior ethmoidal veins. The hæmorrhage may be profuse and fatal. Kelly points to the distinguishing feature of the anterior ethmoidal veins, viz., their close connection with the intracranial circulation, which would explain the profuse and prolonged epistaxis in these cases.

In some cases dependent on general conditions, *e.g.*, plethora, portal congestion, or renal disease, the loss of blood is beneficial within limits, but when it is persistent or profuse it is necessary to check it.

Treatment.—In epistaxis, if the simpler domestic methods have failed, the bleeding point should be sought for. If in the anterior part of the septum, simple compression of the alæ with the finger and thumb may suffice to stop it, or the bleeding point may be touched with the galvano-cautery at a black heat. Kelly finds that epistaxis from the region of the anterior ethmoidal vessels is easily checked by firmly packing between the septum and anterior half of the middle turbinal a strip of gauze reaching to the roof of the nose, or as near it as possible; the parts below may be left free for respiration.

For a less-defined or a more deeply-seated source of hæmorrhage, cold or iced salt and water douches may be tried, or a spray of hazeline. For general oozing, an insufflation of some astringent preparation, *e.g.*, powdered tannic and gallic acid in equal parts, is effectual. When other means have failed the nose should be plugged. For this purpose the best thing is Cooper Rose's inflating plug; Bellocq's sound is very convenient, or a soft rubber catheter can be passed through the nasal passages till it appears in the pharynx, when it is seized with forceps, drawn into the mouth, and the post-nasal plug of antiseptic absorbent wool or lint (about the size of a walnut) is tied on. The plug is then drawn into place till it has occluded the posterior orifice of the nasal fossa on the corresponding side, and the plugging is completed from the front in the usual manner. No plug should be allowed to remain in the nose for more than twenty-four hours. It always gets foul, and if necessary to continue it, it should be removed and the nose, after being cleansed, re-plugged.

HÆMATOMA AND ABSCESS.

Following injuries to the nose we sometimes find, on either side of the septum, a dark red or purplish swelling of fluid blood effused beneath the mucous membrane. In course of time the blood may undergo absorption or organisation, or it may degenerate, forming an abscess, which may become very chronic.

It is well to be cautious in promising that no external

deformity will ensue, for in course of time, when the inflammation and the effused blood becomes absorbed, the nose may develop an unsightly depression near the tip, which may not have been present shortly after the injury was inflicted.

A blood-tumour if small may become absorbed, but if large the blood should be evacuated and the nose kept aseptic by suitable sprays or lotions. Abscess of the septum should of course be evacuated, and appropriate after-treatment adopted.

FRACTURES, INJURIES, ETC.

The nasal septum is mainly formed by the triangular cartilage, the perpendicular plate of the ethmoid, and the vomer: any of these structures may become fractured or dislocated, according to the nature and severity of the injury; generally due to a blow, a fall, or the entry of some sharp-pointed body.

The cartilaginous septum is the most liable to dislocation or fracture, with consequent lateral or downward displacement. Epistaxis generally occurs, and hæmatoma very often. The latter is nearly always bilateral, the blood tumour communicating through the fissure of the fracture. The symptoms of displacement, *e.g.*, nasal obstruction, may only become obvious after the concomitant injury and swelling have disappeared. Hæmatoma and its ultimate consequences have been referred to already.

Fractures of the ethmoid plate, fortunately rare, are apt to be grave, because it is seldom caused, except by violence so considerable that not only does the external bony framework of the nose suffer, but the cribriform plate is often fractured too. Such injuries involving fracture of the base of the skull are naturally very liable to be fatal.

The vomer is rarely fractured, the chief symptoms being epistaxis and septal displacement low down and far back.

Treatment.—The septal fragment, if displaced, should be restored by introducing broad blunt forceps, one blade into each nasal passage, and carefully manipulating; or, when necessary, depressed fragments may be raised by a blunt probe, or sound, or by a little finger in the nasal passage. To retain the fragments in position, Mayer's or some suitable intra-nasal plug, or a piece of thick rubber tubing, may be required till union occurs. For external fractures and their treatment the reader will refer to text-books of general surgery.

CHAPTER XVII.

NASAL NEUROSES.

OLFACTORY, SENSORY, VASO-MOTOR, AND REFLEX NEUROSES.

OCULAR AFFECTIONS DUE TO NASAL DISEASES.

OLFACTORY NEUROSES.

ANOSMIA AND HYPOSMIA.—For clinical convenience it is usual to include under the terms *anosmia*, persistent loss of smell, and *hyposmia*, partial loss, from whatever cause, whether due to local conditions of the mucous membrane or to impairment of the peripheral or central nerve structures. For the proper perception of odours it is essential that the odoriferous particles be able to reach the mucous membrane of the upper part of the nasal passages, and that these should be in a moist condition. Therefore any local affections preventing respiration through the nasal passages, or deposits of mucus and secretion on the olfactory mucous membrane, congenital absence of pigment in olfactory nerve cells, as in albinos, or a permanently dry condition of the membrane, will interfere with or completely abrogate the sense of smell. Acute and chronic catarrh, and mucous polypi, are common causes of anosmia.

The terminal filaments of the olfactory nerves may be so altered or impaired, either from a chronic local inflammatory affection, or in atrophic rhinitis, or as the result of injecting very irritating sprays or douches, that anosmia results. Injuries to the nerves from blows, fractures of the cribriform plate, peripheral neuritis in infectious diseases, particularly influenza, are among the peripheral causes of anosmia.

Central nerve lesions may cause anosmia; thus it may occur in hysteria, locomotor ataxia, general paralysis, or from basilar meningitis, syphilis, aneurysms, exostoses affecting the olfactory bulbs. Intracranial tumours implicating the olfactory lobes and

growths destroying the cortical centres for olfaction or their nerve fibres to the lobes, particularly lesions of the horn of Ammon, the hippocampus and the island of Reil (Lermoyez) are other causes, but it is only very rarely that tumours or intracranial hæmorrhages produce anosmia, as it is unlikely that both bulbs would be destroyed. A few cases of unilateral anosmia, *hemi-anosmia*, in hysteria and from unilateral destruction of the olfactory bulb are recorded.

Reflex anosmia has been observed to follow removal of both ovaries. The term "essential anosmia" has been applied to cases in which no local cause or lesion whatever is present to cause it. It is then usually associated with anæsthesia of the nasal mucosa.

HYPEROSMIA.—Increased sensitiveness to smell is sometimes found in hysteria, neurasthenia, and hypochondriasis, or it may be due to irritation of the olfactory lobes from various causes.

PAROSMIA, or perversion of the sense of smell with imaginary or subjective perception of odours, is nearly always central, and may occur in hysteria, hypochondriasis, in epileptics, in lesions of the anterior lobes, and in insanity.

CACOSMIA, the perception of a bad odour, though generally objective and due to a foreign body in the nose or to accessory sinus disease, may be subjective or functional.

Patients with unilateral anosmia do not generally perceive the loss of power of smell, and when it is bilateral they mostly complain of loss of taste. In testing the sense of smell it is important to use an odoriferous substance which is non-irritating to the nerves of ordinary sensation, and it should be some well-known smell, such as musk, rose, etc.

The *Prognosis* in anosmia depends (*a*) on the nature of the lesion, and (*b*) on its duration. When due to some cause which can be removed, such as polypi, or to hysteria or to peripheral neuritis, the prognosis is favourable provided it has not persisted for a long time. But when the cause is some central lesion or a destructive local lesion, recovery is improbable.

Treatment consists in the removal of the local cause when possible. Local injections of strychnine, local galvanisation, and the administration of arsenic and strychnine, and general treatment of any neurosis is the most that can be done.

SENSORY, VASO-MOTOR AND REFLEX NEUROSES.

We have already noted the anatomical association of the nasal nerve supply (see p. 24) with the vagus nuclei in the bulb and with the sympathetic, and there is ample evidence also of a close relationship between the nasal passages and the sexual organs, to which fuller reference will be made; hence a number of affections are instances of true nasal neuroses. Nevertheless, it is important to avoid referring neuroses to a nasal source whenever it is possible to detect some slight anatomical defect in the nose or to excite a physiological reflex in response to local irritation by a probe. Each case in which an intra-nasal cause for an existing neurosis is suspected must be judged on its merits, and, without unduly underestimating any local lesion, it is well to bear in mind that the victim of neuroses of any kind is apt to lead the unwary practitioner astray and to build high hopes on the supposed discovery of the cause of his protean malady, often ending in bitter disappointment.

On the other hand, in many cases which at first sight appear to be pure neuroses, further careful search will reveal an adequate local cause for the condition of the patient, the removal of which will alone effect relief. Indeed, inasmuch as the nasal mucosa is a highly sensitive area which is in health the region for excitation of numerous physiological reflexes, it is inevitable that correlated reflex areas should sometimes be pathologically excited through the nose, in accordance with the law of irradiation of reflex action, *viz.*, that reflex action extends from nervous areas in which it first operated to neighbouring efferent nerve areas by means of the communications between the different groups of ganglionic nerve cells.

Thus reflex nasal neuroses most frequently excite physiological reflexes and other symptoms in the upper respiratory tract—*e.g.*, sneezing, coryza and vascular turgescence; next in frequency, morbid reflex phenomena in the lower tract—*e.g.*, asthma, vasomotor bronchitis, while only very rarely are epilepsy, melancholia, cardiac symptoms, etc., dependent on nasal sources.

HYPERÆSTHESIA is generally the immediate local factor in nasal cough, paroxysmal sneezing, hay fever, and hay asthma, but in all these conditions it is usual to find more or less definite abnormal conditions of the nasal mucous membrane associated

with hyperæsthesia at one or the other of the sensitive spots in the nasal passages.

Nasal cough is usually a hard persistent cough, generally ceasing during sleep, and unattended with expectoration or any complication.

Neuralgia in the various branches of the fifth nerve may be reflex, or may occur in *tic douloureux*.

ANÆSTHESIA from implication of the fifth nerve may be due to hysteria, cerebral tumours, or intra-cranial syphilis. Incomplete anæsthesia is not unusual in old-standing large polypi and in various chronic degenerative diseases of the mucous membrane.

VASO-MOTOR RHINITIS.

Simple erectile swelling and vascular engorgement occur in two forms: (*a*,) Periodic vascular swelling; (*b*,) Vascular engorgement with coryza and sneezing. Neurotic persons, especially females, gouty subjects, and excessive alcoholic drinkers, are prone to suffer from these phenomena (*see Plate IX, Fig. 5*). It is generally an indication of nervous prostration, imperfect digestion, or of sexual excesses. J. N. Mackenzie had a patient who invariably suffered from coryza after sexual indulgence. It is sometimes associated with slight abnormalities in the nasal passages.

The fulness of the nasal mucosa may be accompanied by general redness of the nose, or of the face, while in rare instances, the redness and swelling of the nose which come on in heated rooms or after meals, are due to pressure of the distended middle turbinated bodies on the septum. Of this condition I have seen one or two instances, and the periodical redness and swelling of the nose, which rendered the patients almost unfit for society, were completely relieved by cauterising the middle turbinated bodies.

Treatment consists in attention to the general health, while, *if necessary* only, the galvano-cautery may be used, one or two linear cauteries being made over the turbinated bodies or septum.

CORYZA OEDEMATOSA is closely allied to rhinal hydrorrhœa. Mullhall records six cases to which he applies this term. It

consists of a serous infiltration into the connective tissue of the inferior and middle turbinals, which is sometimes migratory and appears to be associated with irregularities of indigestion in neurotic subjects. It is not an inflammatory affection, but is more allied to Quincke's œdema.

RHINORRHOEA. Two distinct affections are characterised by the escape of clear watery fluid from the nose, viz. :—

(1.) *Rhinal Hydrorrhœa* a vaso-motor neurosis, with transudation of watery fluid from the nasal mucous membrane, and

(2.) *Cerebro-spinal Rhinorrhœa*, in which there is an escape of cerebro-spinal fluid from the roof of the nasal fossæ. It is in no sense a nasal neurosis, but nevertheless is most conveniently described here also.

RHINAL HYDRORRHOEA.

(*Coryza vasomotoria periodica.*)

Idiopathic Rhinorrhœa, an affection of which the essential feature is profuse watery discharge from the nose, as distinguished from the escape of cerebral spinal fluid through the nose; while in some cases the discharge of fluid is preceded or accompanied by sneezing and lachrymation, itching or pricking sensation in the nose; in others the "running" from the nose is the one and only symptom.

The *causes* of the affection are generally indefinite, the patients are usually neurotic, highly strung or overworked, and in some cases exposure to cold has seemed an exciting cause. It is probable that the fluid is a true secretion rather than a mechanical exudation, and due to excitation of the secretory filaments derived from the sphenomaxillary ganglion.

After the rhinorrhœa has persisted for some hours the mucous membrane becomes swollen and œdematous. As much as two or three quarts have been known to come away in the twenty-four hours. The discharge is a straw-coloured fluid consisting of water, with traces of sodium chloride and mucus, and does not reduce Fehling's solution.

For the differential diagnosis between rhinal hydrorrhœa and cerebro-spinal rhinorrhœa, see p. 352.

CEREBRO-SPINAL RHINORRHOEA.

Spontaneous discharge of cerebro-spinal fluid through the nose has been termed cerebro-spinal rhinorrhœa by St. Clair Thomson, to whose work on the subject I am mainly indebted for the following description of the affection. In one case the escape of fluid was found *post mortem* to have taken place through a small hole in the dura mater by the side of the crista galli; in two others examined *post mortem* the point of exit could not be found, but it is probable that the escape usually takes place through the cribriform plate of the ethmoid. Generally there is a history of headache or other mental symptoms, which are relieved with the escape of cerebro-spinal fluid.

The discharge of fluid is generally from one nostril, and it persists night and day without much intermission for weeks, months, or years. When the intermissions do occur, the cerebral symptoms, headache, etc., recur or are aggravated. There is no accompanying lachrymation or photobia, but the long persistence of the flow may lead to the formation of nasal polypus.

Cases of spontaneous discharge of cerebro-spinal fluid from the nose have also been recorded by Scheppegegrell, Leber, Körner, and Freudenthal.

Thompson's case was that of a female, aged 25, in whom the escape of fluid from the left nostril persisted day and night for over three years. She used from five to six handkerchiefs daily. There were four intermissions in the flow of sixteen days, twenty-eight days (two occasions), and two months' duration respectively; at these times very severe headaches were present, chiefly over the left eye and back of the head, though at other times the health was excellent.

Freudenthal's case was that of a female, aged 50. In 1898 she had high fever, and since that time up to time of reporting (18 months), the nose had been steadily dripping. A year before the dripping began she complained of pain in the forehead above the bridge of the nose, and she thought she was going out of her mind. Her mental obscurity, which had been pronounced, was relieved when the dripping came on, but the mental symptoms were aggravated again when for a time the dripping grew less.

Diagnosis.—The differential diagnosis between cerebro-spinal rhinorrhœa and nasal hydrorrhœa depends mainly on the chemical composition of the escaping fluid, though the character of the symptoms also gives some indication of the nature of the diseases in question. Professor Halliburton's analyses (cited by Thomson) may be contrasted as follows:—

CEREBRO-SPINAL FLUID.

Fluid is perfectly transparent like water, and contains no sediment. It is not viscous. It is faintly alkaline in reaction, and either tasteless or slightly salt.

It gives no precipitate (mucin) on adding acetic acid.

The sp. gr. is 1005 to 1010.

Cold nitric acid gives a precipitate which disappears on heating.

Saturation with magnesium sulphate or sodium chloride gives a precipitate.

On boiling, there is not more than a trace of coagulum of serum globulin and serum albumin. *When boiled with Fehling's solution there is a reduction of copper* (due to pyrocatechin or some similar body).

The reducing substance may be obtained in needle-like crystals by evaporating to dryness an alcoholic extract of the fluid.

The symptoms also may be contrasted :—

CEREBRO-SPINAL FLUID FLOW.

Headaches precede or follow the flow, but cease or are diminished during the persistence of flow.

No nasal symptoms.

It persists night and day.

The fluid drying, leaves the handkerchief pliable.

No treatment is admissible.

NASAL HYDRORRHOEA FLUID.

Fluid is thick and viscid, and slightly opalescent.

Histological examination shows the presence of amorphous matter and mucous corpuscles.

It gives, with acetic acid, a stringy precipitate (mucin).

On boiling this precipitate with dilute sulphuric acid, a reducing, sugar-like material is formed. This is also characteristic of mucin.

The fluid contains a small amount of proteid coagulable by heat; *it does not reduce Fehling's solution*. Proteoses and peptone are absent.

The presence of mucin and absence of reducing substance, as well as the considerable percentage of proteids and solids, are quite sufficient to distinguish this fluid from normal cerebro-spinal fluid.

NASAL HYDRORRHOEA.

Feeling of malaise sets in with the discharge, and disappears with its cessation.

Often ushered in with sneezing, lachrymation, etc.

It rarely continues during sleep.

The fluid drying, leaves the handkerchief stiff.

Disease is amenable to treatment.

Treatment of Vaso-motor Neuroses.—The treatment mainly consists in attention to the neurotic condition of the patient, by massage, baths, change of air, tonics, etc. Dyspepsia, constipation, or any irregularities in the alimentary tract require

appropriate treatment. Engorgement or œdematous swelling of the turbinated bodies may be reduced *when necessary* by linear scarifications, or by one or two linear cauterisations with the galvano-cautery. Rhinal hydrorrhœa has yielded to treatment with small, repeated hypodermic injections of atropine and strychnine. Atropine may be injected into the nasal mucosa. The galvanic current also appears to have some curative influence, one pole being applied directly to the nasal mucous membrane.

PAROXYSMAL SNEEZING AND HAY-FEVER.

Etiology and Pathology.—Paroxysmal sneezing may be due (*a*) to reflex peripheral irritation, as in the earlier and milder forms of true hay-fever, in which the symptoms appear only when the patient is exposed to pollen, etc., and in cases due to some local abnormality in the nose; or (*b*) to a central neurosis.

Paroxysmal sneezing has often been noted in connection with sexual excitement.

Exciting causes.—Paroxysmal sneezing may be excited by any irritation of the fifth nerve, either directly, *e.g.*, from irritating dust in the nose, impinging on a sensitive area, or from the irritation due to a septal spur, or reflexly by the action of bright sunlight (sensori-motor), or by sexual causes, or by the imagination (ideo-motor), as for instance the occurrence of sneezing and a regular attack of hay-fever induced in a susceptible person by the sight of an artificial rose in the room.

Hay-fever is simply paroxysmal sneezing set up by particular forms of irritating dust, to wit, pollen grains. Attention was first seriously directed to hay-fever by Bostock in 1819, who endeavoured to prove that the symptoms were due to the solar rays; but the researches of Blackley



FIG. 175.
Two grasses commonly causing Hay-Fever.

have proved that it is due to the action on the mucous membrane of the pollen grains of certain natural orders of plants, especially the pollen of Gramineæ, *anthoxanthum odoratum*, meadow grass, barley, wheat

and oats. Roses, and in America, Roman wormwood, ragweed, etc., have also a peculiar tendency to excite this affection. Any form of dust impinging on the nasal mucosa in susceptible individuals, may bring on the symptoms.

Predisposing causes.—As the exciting causes of hay-fever are practically universal, and every one must be exposed to them, while comparatively few suffer, it is obvious that individual predisposition is necessary for the symptoms to appear. And this applies to all cases of paroxysmal sneezing which generally occurs in dwellers in cities, in the educated classes, and especially in those persons of the neurotic temperament; therefore to some extent it is hereditary.

Local Conditions.—The exciting causes are universal, the predisposing are very common, while the affection itself is *relatively* rare. Thus, a third factor is generally necessary, and this is found in the abnormalities and morbid conditions of the nasal passages. We are largely indebted to Roe, of New York, and Daly, of Pittsburgh, for the recognition of these local conditions as causes of hay-fever, viz.: (1,) hypertrophic rhinitis; (2,) spurs and bony projections of the turbinals or septum; (3,) septal deviations; (4,) polypi and adenoid hypertrophy of the naso-pharynx; (5,) peculiarly sensitive areas.

Thus, for the occurrence of paroxysmal sneezing, three factors are generally necessary:—

- (1,) The predisposing constitutional condition;
- (2,) An external irritant;
- (3,) A pathological condition of the nasal mucous membrane.

Symptoms.—Some neurotic women are subject to attacks of sneezing, occurring in paroxysms of thirty or forty sneezes, especially on rising in the morning, generally attended with rhinorrhœa, lachrymation, and temporary nasal obstruction, and sometimes with pain over the bridge of the nose. I have recently had a male patient who sneezed two hundred times a day, and was completely prostrated by the affection; it was associated with a septal spur pressing on one of the inferior turbinals.

The symptoms of hay-fever come on in Great Britain about the middle of June, and in America about the middle of August, while simple paroxysmal sneezing may occur at any period. At first only a slight itching of the inner canthus is observed,

and watering of the eyes. In a day or two some irritation in the nose with watery discharge comes on, with nasal obstruction, due, in fact, to vaso-motor rhinitis, and pricking and dryness in the throat. Very soon attacks of sneezing supervene, and recur with increasing frequency without the pleasant sense of relief that is usually associated with a good sneeze. The conjunctivæ become injected, the eyes bloodshot, and the nasal passages more or less blocked up by the swelling of the mucous membrane and turgid erectile tissues. The fauces become relaxed, and even ordinary conversation becomes an effort.

At first the symptoms are merely annoying, but in pronounced cases with each annual recurrence they become more and more severe, and life is rendered a perfect misery during the three brightest months of the year. The symptoms are associated with intense prostration, altogether out of proportion to the local irritation, and health is greatly impaired for many weeks after the peculiar symptoms have subsided. A good many victims to hay-fever outgrow their susceptibility in later life.

Treatment.—As in all neuroses, we should bear in mind the importance of treating the general constitutional condition by promoting the health and strength. For this purpose a general hygienic and tonic treatment should be adopted, and in hay-fever a tonic course is desirable for a few months before the usual time for the symptoms to come on.

Idiopathic paroxysmal sneezing dependent on functional affections of the nerve centre should be combated by nervine tonics, such as arsenic, strychnine, valerianate of zinc, and quinine.

If sneezing is due to abnormal conditions of the nasal passages, appropriate measures should be undertaken with a view to their removal. When erectile swelling and vascular injection of the mucous membrane is the only abnormality, I have found that spraying the nasal passages with a solution of biniodide of mercury (strength 1 in 50 to 1 in 20) has proved highly successful in curing many cases, myself among the number. A cocaine spray should be used beforehand, but as the cocaine is destroyed by the mercurial salt, it is necessary to relieve the pain which soon comes on by a hypodermic injection of morphine. The mucous membrane of the nose becomes much congested and swollen. In about three hours the pain and

swelling subside, and are followed by a nasal catarrh lasting two or three days. In suitable cases if this be efficiently done at the onset of the symptoms of hay-fever, the patient will remain free throughout the season, and there are very few people who have suffered from hay-fever who will not readily undergo this or any treatment which promises relief. This method has the advantage of leaving the sense of smell unimpaired, and involves no destruction of tissue. Sir Andrew Clark advised a somewhat similar procedure for hay-fever, which gave relief in about 50 per cent. of his cases, viz., painting the nasal mucous membrane with a mixture of carbolic acid, quinine, and perchloride of mercury.

Hypertrophic rhinitis and sensitive spots should be cauterised with the galvano-cautery, while septal deviations, polypi, spurs, and other abnormalities should be removed.

I have used with some success in mild cases of hay-fever insufflations of a powder composed of adrenal capsule 1 part, boracic acid 2 parts, and orthoform 3 parts. Cocaine should never be locally applied for the relief of hay-fever; it only tends to aggravate the condition after its transient good effects have passed off. Inhalations of the vapor benzoini, or v. benzoini c. chloroformo, are very soothing, as also are creasote, camphor, pinol, terebene, etc. These latter, dissolved in liquid vaseline, may be sprayed into the nostrils.

Treatment by hypodermic injections of aqueous solutions of plants or flowers which cause hay-fever has been advocated by Holbrook Curtis, and he claims to have obtained gratifying results from repeated injections of extract of ragweed and other flowers, with a view to the production of immunity to their influence.

ASTHMA.

It has long been recognised that asthma may be associated with intra-nasal disease, but it is only since Voltolini's classical case of asthma, which he cured by the removal of nasal polypi, that serious attention has been directed to nasal abnormalities as a cause of asthma.

The anatomical connection between the bulbar nuclei of the fifth nerve and the vagus, by means of which the upper and lower respiratory tracts are physiologically associated, have already been described and figured (pp. 24, 25), and we can therefore readily comprehend why in many cases there seems such close interdependence in their morbid relationship.

Asthmatic attacks are generally preceded or accompanied by sneezing and rhinorrhœa, or may alternate with, replace or be replaced by paroxysmal sneezing, in fact these nasal phenomena must be regarded as part of the asthmatic syndrome, by no means indicating that the bronchial symptoms are dependent on the nasal. Just as bronchitis, emphysema, and other pathological conditions are a usual consequence of frequently-recurring attacks in chronic asthmatics, so we not unnaturally observe as a consequence of the nasal phenomena of asthma, hypertrophic rhinitis, water-logged mucous membrane, and perhaps even mucous polypi, these nasal affections being often the consequence, not the cause, of asthma.

However, when these or other intra-nasal affections cause more or less interference with nasal respiration, the bronchi are more exposed to the influence of irritating particles or exhalations by buccal respiration, and in these cases restoration of the nasal respiratory functions by appropriate treatment will often tend to keep off the asthmatic attacks.

What are the nasal affections which may require treatment in asthma? The most hopeful cases for intra-nasal treatment are those in which mucous polypi are found, and it is more usual for small polypi to occur in asthma than for large polypi, which, though causing more complete stenosis, are less mobile, and therefore probably less likely to irritate the neighbouring mucosa. On the tuberculum septi a spot of hypertrophied or thickened soft mucous membrane or a septal spur will sometimes be found impinging on the corresponding turbinal. In others, we shall find that there are particularly sensitive areas on the nasal mucosa, which, when probed, give rise to violent cough; I do not allude to the simple cough reflex obtained on probing many normal noses, and which may also be excited in many persons by a cotton-wool probe or plug in the external auditory meatus.

But often we shall discover nothing beyond hypertrophic rhinitis, œdematous mucous membrane, or vascular engorgement of the turbinal bodies. By the appropriate treatment of these and other intranasal conditions, I have obtained successful results; many cases have been immensely relieved and some cured. Speaking generally, I have found that cases of inherited asthma are much less frequently due to nasal causes than the acquired

forms, and it cannot be too strongly emphasised that the existence of any of these nasal abnormalities in an asthmatic patient does not necessarily imply that they are causal factors. Yet if on the application of cocaine solution to the nasal mucous membrane the asthmatic attack is aborted or obviously relieved, we are justified in saying to the patient "it is highly probable that local treatment of the intranasal abnormalities will result in considerable relief and possibly a cure, but whether the relief will be of long-standing or the cure permanent cannot be foretold; it is not even possible to promise that this local treatment will exercise any beneficial effect on the cause of the asthma."

The treatment of the various nasal conditions alluded to has already been described elsewhere, but the general treatment of the patient should in all cases receive careful attention.

ÉPILEPSY.

Occasionally epilepsy has seemed to be caused by nasal abnormalities, the removal of which has been followed by cessation of the epileptic seizures, and I have had fairly definite proof that epilepsy may be induced by intranasal irritation in a patient who had an ordinary epileptic fit while I was cauterising the nose for polypi. He never had a fit before, and had no further attack, at any rate during the next eight years.

Other similar cases have been recorded by different observers.

Baron reports two cases, one a case of nasal polypus, removal of which was followed by marked alleviation of the epileptic seizures; the other, a young unmarried woman, who had epileptic fits at her menstrual periods from the time menstruation began. Her inferior turbinated bodies were greatly hypertrophied, and she was always more troubled with nasal stenosis during the menstrual periods, and it was at that time only that the fits occurred. Removal of the hypertrophied tissue was followed by cessation of the fits for seven or eight months, and when they re-appeared the turbinal hypertrophy was found to have recurred.

CARDIAC AFFECTIONS.

The effect of irritation of the nasal mucosa upon the movements of the heart and pulse have been studied by Guder. He tested in all forty-three subjects, thirteen with normal noses and thirty with nasal disease, and repeated his tests on several occasions in each. He found that irritation of the nasal mucosa by the probe, the galvano-cautery, irritating insufflations, etc., was entirely negative.

I have never seen any instance of reflex influence on the heart from nasal disease, but Spencer Watson records a case of tachycardia which was associated with, and apparently due to, nasal polypi. Charsley observed temporary exophthalmos with tachycardia, the pulse ranging as high as 110 per minute, coming on and lasting for a period of three months after the galvano-cauterisation of one of the inferior turbinals. Symptoms of Graves's disease have been attributed to nasal disease; thus Hack, in a case associated with chronic rhinitis, found that the goitre and tachycardia vanished after treatment of the rhinitis, and B. Fränkel and Hopmann report similar cases cured by nasal treatment.

OCULAR AFFECTIONS DUE TO NASAL DISEASE.

The lachrymal duct forms a direct communication between the eye and the inferior meatus of the nose, and is lined by mucous membrane continuous with the ocular conjunctiva above and the nasal mucosa below.

But there is also a normal reflex connection between the eye and the nose, and probing of the nasal passages will usually excite lachrymation and hyperæmic injection of the conjunctiva, while conversely bright light will often cause sneezing.

Further, the nasal passages have other important anatomical relations with the orbital cavity and its lachrymal duct, the frontal sinuses forming in part the roof of the orbit, the ethmoidal cells the inner wall, and the maxillary antrum the floor, while the anterior ethmoidal cells surround more or less completely a portion of the nasal or lachrymal duct. Another accessory nasal cavity—the sphenoidal sinus—is in relation with the optic nerve and the ophthalmic artery.

Nasal and ocular affections may thus be produced: (*a*.) by direct extension along the lachrymal duct; (*b*.) directly through the upper, inner, or lower wall of the orbit, or by extension of disease from the sphenoidal sinus; (*c*.) reflexly.

Epiphora and dacryocystitis are not infrequently due to nasal disease causing occlusion of the opening of the nasal duct in the inferior meatus, though doubtless in some cases the disease is primarily an ocular affection which spreads to the nose. I have seen cases of unilateral lupus or hypertrophy of the inferior turbinal cause epiphora. Lupus may extend to the eye from the nose, and this, Seifert states, is not so rare as is generally supposed. Berger found that chronic hypertrophic rhinitis is the chief source of ocular troubles depending on the nose. Seifert states that in thirty-eight cases of dacryocystitis reported

by Gruhn, nasal disease occurred in thirty-six, whilst of forty-eight cases of disease of the lachrymal apparatus, reported by Gluck, all exhibited nasal abnormalities.

The ocular complications of nasal accessory sinus disease are discussed in connection with sinus disease. Of reflex conditions, strabismus, narrowing of the visual fields, photophobia, myopia, scintillating scotoma (Hack), asthenopia, epiphora, lachrymation, œdema of the lids, blepharospasm, and exophthalmos have been reported.

Laurens records two cases, one of a male, aged 31, in whom an intra-nasal operation for the removal of synechie between the inferior turbinal and the septum was followed by sudden and permanent relief to blepharospasm of some months' duration; the other case was a girl, aged 6, in whom left convergent strabismus was said to have disappeared a few days after an operation for the removal of adenoids. In a case of Cresswell Baber's, diplopia due to paresis of the right external rectus developed after removal of polypi with the cold snare; the ocular symptoms disappeared in about six weeks. Numerous small growths were subsequently removed without return of ocular symptoms, but the patient had had a similar attack when operated on with forceps two years previously. Mention has already been made of cases of reflex exophthalmos associated with nasal affections.

CHAPTER XVIII.

DISEASES OF THE ACCESSORY SINUSES OF THE NOSE.

INTRODUCTION—ACUTE SINUSITIS—CHRONIC EMPYEMA OF ACCESSORY SINUSES.

GENERAL INTRODUCTION.

DISEASES of the nasal accessory sinuses are of great clinical importance, for they constitute a large proportion of the nasal affections which come under the notice of medical practitioners. They are often very difficult to diagnose and treat, and may be the cause of great inconvenience to the patient and not rarely undermine the health or lead to fatal complications. One assumes a knowledge of their anatomy (see p. 8 *et seq.*), without which it is difficult, if not impossible, to appreciate the symptoms and signs of inflammatory disease in these sinuses.

Much of the difficulty surrounding the subject under discussion disappears if we consider these accessory sinuses simply as a single series of cavities surrounding the nasal passages into which they open. They are all liable to inflammation by extension from the nasal passages and other causes, and in pathology, symptoms and general principles of treatment they have much in common, the differences being chiefly due to their different anatomical position and relations.

From the accompanying diagram it will be seen:—

(1,) That the accessory sinuses may be divided into two groups: (*a*) the *anterior group*, comprising those whose ostia open into the middle meatus, that is, below the oblique line of attachment of the middle spongy bone (indicated by a thick line), and (*b*) the *posterior group*, comprising the posterior ethmoidal cells and sphenoidal sinuses, which open above the middle turbinal in the superior meatus.

(2,) That pus discharging from any of the anterior group will appear in the middle meatus beneath the middle turbinated body, and that it will tend to pass towards the anterior nasal orifice, especially on stooping; while pus coming from any of the posterior group appears above the middle turbinal, that is, in the olfactory fissure, and it not only appears far back, but running down is further directed towards the posterior nares by the middle turbinal, and therefore passes into the rhino-pharynx. The pus from this posterior group can often be seen on posterior rhinoscopy, coming from above the posterior extremity of the middle turbinal.



FIG. 176.

Diagram to show the arrangement of the accessory sinuses and their apertures of communication with the nasal passages. The dark oblique line indicates the attachment of the middle turbinated bone, dividing the various cavities into A, the anterior group, and P, the posterior group. The anterior group comprises F, the frontal sinus A E, the anterior ethmoidal cells, and the maxillary sinus, O M, being the ostium maxillare. The anterior ethmoidal cells are further divisible into (3) lower, AE', (2) middle, AE'', and (1) AE''', upper cells: 1, the infundibulum. The posterior group comprises, P E, the posterior ethmoidal cells, and S, the sphenoidal sinus.

(3,) That suppuration arising in the frontal sinus or anterior ethmoidal cells must often spread from one to the other, and that the pus in its downward course from these cavities may find its way into the maxillary sinus. On the other hand, owing to

the presence of polypi, or even from capillary attraction in the infundibulum, pus escaping from the antrum may find its way to the frontal sinus and ethmoidal cells and so infect them. Thus empyemas of the different cells in the anterior group often co-exist.

(4.) That, for similar reasons, empyema of the sphenoidal sinus and posterior ethmoidal cells tend to be associated.

(5.) That pus from the frontal sinus and the anterior ethmoidal cells tend to discharge more freely in the upright position, and to be a continuous discharge; pus from the antrum of Highmore will tend to accumulate in the upright position and to flow out freely from the *ostium maxillare* on lowering the head; pus from the sphenoidal sinus will also tend to accumulate in the upright position and to flow more freely on stooping; while pus from the posterior ethmoidal cells is not likely to be large in quantity but escapes continually.

(6.) That in closed empyema, in which the pus cannot escape, (*a*) distension of the frontal sinus may cause bulging of the posterior wall into the cranial cavity, bulging of the anterior wall above the orbit, bulging of the floor (see *Plate XXX*), causing displacement of the eyeball downwards and outwards; (*b*) distension of the anterior ethmoidal cells may cause bulging of the roof of the cells just outside the cribriform plate (see *Plate XXVI*), or, as more commonly happens, bulging of cell walls into the orbit, causing outward and downward displacement of the eyeball; (*c*) distension of the maxillary sinus may cause bulging of the cheek, the inner wall of the nose, or of the hard palate (see *Plate XXI*); (*d*) distension of the sphenoidal sinus may cause compression of the optic nerve, cavernous sinus or ptosis and strabismus from compression of nerves in the sphenoidal fissure (see *Plate XXVI*).

With these facts in mind, it is comparatively easy to understand the symptoms and signs of sinusitis and their relative value in arriving at a differential diagnosis. Nevertheless, it is sometimes a very difficult matter to say for certain what particular sinus is implicated.

Inflammation of the sinuses may be acute or chronic, and the secretions catarrhal or purulent.

If the ostium of the sinus remains more or less open, it is termed *an open sinusitis*, but if the aperture is occluded we have

what is called a closed sinusitis, and when the secretion is catarrhal it is termed a "mucocoele"; if purulent, a "closed empyema." In other cases the secretion is able to escape, and is unattended with local symptoms; to these the term "latent" is applied. Dmochowski, E. Fränkel, and Zuckerkandl, found, *post mortem*, a considerable percentage of cases of maxillary sinus disease in which there had been no sign of its existence during life.

The division of empyemas of the nasal sinuses into "open" and "closed" is convenient for clinical purposes, and yet often enough a case cannot be placed in either group. For instance, an empyema may alternate between the two conditions, the ostium being more or less closed with aggravation of the symptoms, till the accumulating secretion forces an exit with accompanying relief of many of the symptoms, and, from being a closed empyema, it becomes an open empyema. Thus the division of empyemas into *closed*, *alternating*, and *open*, is more satisfactory for clinical purposes.

Etiology.—The accessory sinuses being lined by mucous membrane in continuity with that of the nose, they are liable to participate in all acute inflammatory affections of the nasal passages by direct extension, and to be secondarily infected by various chronic diseases, or by the secretions escaping from one sinus finding its way into neighbouring accessory sinuses.

To avoid repetition the various causes of inflammatory processes in the different sinuses may be grouped together thus:—

(1,) Simple acute rhinitis may involve the mucosa lining any of the accessory sinuses, and chronic catarrhal or purulent sinusitis may result. The accessory cavities do not invariably participate in inflammatory diseases of the nasal passages, as some are inclined to think. In the course of his anatomical researches on the nose, Harke not unfrequently found the mucous membrane of the nasal passages very severely inflamed, while that of the accessory cavities was entirely normal.

(2,) Acute infectious diseases, especially influenza and septic infection (erysipelas, etc.) less frequently measles, scarlatina, typhoid fever, small-pox, pneumonia (v. Besser), diphtheria, glanders, gonorrhœa, and acute rheumatism.

(3.) Chronic infectious diseases, tuberculosis, and syphilis. Pus in the antrum has been known to be caused by the presence of a malignant growth.

(4.) The invasion of a sinus by insects, larvæ, etc., has several times caused a frontal sinusitis. Dochmeins found *anchylostomum duodenale* in one patient's frontal sinus. Harke has found particles of tobacco in the antra of snuff-takers.

(5.) Occlusion of the ostium by mucous polypi. We must bear in mind that polypi and a polypoid condition of the mucous membrane around the apertures is usually the result rather than the cause of empyema. Two cases of empyema of the maxillary sinus following plugging of the nasal fossæ for epistaxis are reported by Saint-Hilaire.

(6.) Pus descending from one sinus and entering or infecting another. Thus in a large percentage of cases antral empyema is the direct result of pus descending from the frontal sinus passing into the antrum; and some cases of obscure origin are probably due to simple catarrhal secretion in excess gaining entrance into the antrum and there undergoing decomposition.

7.) Injuries. Naturally it is the frontal and ethmoidal cells and antrum of Highmore which most often become affected, being most liable to injury from blows on the face and nose. Two cases of antral empyema following section of the infra-orbital nerve are reported by Langenbeck.

(8.) Mercurial ptyalism and lead poisoning may cause antral empyema. Wagner records a case due to chronic lead poisoning in which the mucous membrane of the antrum showed a peculiar blueish-gray hypertrophy, scrapings of which gave a characteristic reaction with sulphide of sodium. Phosphorus workers are liable to inflammatory disease and necrosis in the antrum and ethmoid cells.

9.) Some cases of antral empyema are undoubtedly secondary to caries of the teeth, the sockets of which project into the floor of the antral cavity, but it is very difficult to determine exactly what proportion must be attributed to dental caries inasmuch as there are relatively few cases in which carious teeth are not present, which from their position could have had no causal influence, and there is little reason to doubt that chronic antral empyema is often itself a cause of caries of the teeth corresponding to the affected cavity.

This list of diseases and conditions which have been known to cause sinusitis is a long one, but it will be found that the large majority of cases have succeeded attacks of influenza, and it is since the occurrence of the influenza epidemics commencing in the year 1889 that inflammatory diseases of the accessory sinuses have become common affections.

The bacteriology of nasal sinusitis has been the subject of investigations by Howard and Ingersoll, of Cleveland, and as a result of their own researches in eighteen cases and of very numerous collected observations, they reach the following conclusions: Acute and chronic inflammations of the accessory sinuses are with few exceptions (*aspergillus* and *vermes*) caused by bacteria. The bacteria found are those usually present in the nose in acute and chronic rhinitis, nasal tumours, etc. The most important and usual micro-organisms found in sinusitis are those common agents in the causation of inflammation in other parts of the air passages, viz., *diplococcus lanceolatus*, pyogenic *staphylococci* and *streptococci*, bacilli of the group of Friedlander's bacillus, the bacillus diphtheriae, and the bacillus of influenza. They note the finding by Frankel of inflammation of these sinuses in individuals suffering with chronic diseases such as nephritis, arterio-sclerosis, and lung tuberculosis, which are now proverbially known so to lower the general resistance as to favour secondary infections, as of special interest.

In one of my cases of sphenoidal sinus empyema, the pus withdrawn with aseptic precautions yielded two micro-organisms in an agar culture, viz., a bacillus, with rounded ends, 2-5 μ long, about $\frac{1}{2}\mu$ thick, and a small diplococcus, thought to be the *diplococcus intercellularis meningitidis* by D. S. Davies.

The secretion may be clear mucous fluid, more generally it is greenish or yellowish muco-purulent or purulent. It is often canary-yellow colour due to the presence of *staphylococcus pyogenes aureus*.

In catarrhal inflammations the mucous membrane at the outset is injected, and is often swollen and œdematous. The columnar ciliated epithelium seems to be shed very early, and soon is completely lost. In purulent inflammations the mucous membrane is infiltrated with cells and is swollen, and presents numerous sub-mucous hæmorrhages, with papilliform hypertrophies in old cases, and not rarely a number of polypi will be found growing from it. Hyperostoses and osteophytic processes are described by Zuckerkandl. But more important still than the formative osteitis is the rarefying osteitis with erosion of the wall in some long-standing cases, in consequence of which the wall may become softened or completely perforated with escape of the purulent contents of the frontal, ethmoidal, maxillary, or sphenoidal sinus, as the case may be, into neighbouring tissues or cavities.

ACUTE SINUSITIS.

(Syn. SYRINGITIS*).

Symptoms.—The supra-orbital aching and sense of fulness which is so common in ordinary acute nasal catarrh is due to implication of the frontal sinuses; a sense of fulness and indefinitely localised headache and similar pain and discomfort in the orbit and cheek may be due to sphenoidal sinus catarrh; dental neuralgia arises from acute catarrh of the ethmoidal and maxillary sinuses.

In the vast majority of cases of acute catarrhal inflammation, the symptoms are not pronounced, and subside spontaneously, the catarrhal secretion being discharged into the nasal passages. But sometimes in the acute catarrhal cases, and usually in the rare acute suppurative sinusitis, the swelling of the mucosa blocks the apertures of exit so that the affected sinuses become painfully distended with the exudation.

The pain may be acute and lancinating with an intolerable sense of distension, referred to the frontal region, root of the nose and orbit, or to the cheek, according to the particular sinus involved, and is often associated with localised redness, tenderness and inflammatory swelling. The pain is usually greatly increased by blowing the nose, coughing, sneezing, lowering the head, or by any exertion.

Tenderness and increased pain on pressure in the upper internal angle of the orbit is usual in frontal or ethmoidal sinusitis; over the antrum, or at the exit of the superior maxillary nerve in maxillary sinusitis. Generally in the course of a few hours the distension of the accumulating secretion forces an exit through the natural opening, and with the sudden evacuation of the turbid, greenish, sero-purulent fluid the painful symptoms disappear.

The same course of events may recur several times for one or two days, or, on the other hand, no spontaneous evacuation may take place, in which case the walls of the cavity may yield, and a mucocoele, or, if the secretions become purulent, an empyema, results. Thus bulging of the posterior wall of the frontal sinus may cause cerebral compression, the eyeball may be

* Σύριγξ, a cavity.

displaced in various directions by the frontal, ethmoidal, or maxillary sinus, as in chronic empyema with retention.

Again, the walls of the sinus may be eroded and the contents escape; or, without perforation of the walls, pathogenic organisms may find their way out through vessels or lymph channels, and set up suppurative inflammation. In these ways an acute suppuration of the frontal sinus or ethmoidal cells may cause meningitis, or an extra-dural abscess beneath the frontal lobe of the cerebrum, or suppurative cellulitis in the orbital fossa.

These complications are more liable to arise in the chronic purulent sinusitis, and will be discussed more fully in that connection; but it is necessary to bear in mind that in acute suppurative disease of these sinuses, likewise of the sphenoidal sinus, very grave symptoms pointing to cerebral irritation and compression may rapidly develop.

Treatment.—The main indications are to relieve the pain and to promote evacuation of the secretions through the natural apertures of exit. Hot fomentations externally and inhalations of steam impregnated with tincture of benzoin and chloroform are soothing, and should be combined with the internal administration of phenacetin, phenazone, aconite or belladonna to relieve the pain, headache and feverishness. It is well to avoid alcohol as it often aggravates the disease.

The swelling and obstruction of the natural channels to the sinus may be reduced by spraying the nasal passages, and especially the region of the ostium of the affected sinus, with an oily solution of cocaine and menthol, or an aqueous solution of supra-renal capsule and cocaine. Hartmann's suggestion that Politzer's bag should be used, although it often affords instant relief, does not commend itself to me except perhaps in cases where one maxillary antrum or frontal sinus is alone involved, as there is always a risk in acute inflammatory affections of the upper respiratory tract of driving infective material into the Eustachian tubes. If by these means evacuation of the retained secretion cannot be brought about, and especially if there is reason to suspect that suppuration has occurred, the affected cavity must be opened by operative measures as in chronic empyema, and efficient drainage secured. It is obvious that signs of extension of the inflammation to the orbit or to the cranial cavity are indications for immediate operation.

CHRONIC EMPYEMA OF THE ACCESSORY SINUSES.

CHRONIC EMPYEMA OF THE MAXILLARY ANTRUM.

Chronic empyema of the maxillary antrum, or antrum of Highmore, was described by John Hunter, but the credit of recognising the frequent occurrence of the affection without the classical signs of pain, swelling and tenderness, is due to Ziem, who published his first paper in 1886.

It may occur at any age, though it is rare in children. D'Arcy Power reports its occurrence in a boy eight weeks old, probably caused by forceps in delivery; and Rudaux another instance of empyema in an infant, only three weeks of age, in whom it was set up by a prematurely developed tooth in the antral cavity.

Symptoms.—In the open or latent form, by far the most common, patients generally complain of a constant or periodic discharge of considerable quantities of foetid pus from one side of the nose. Not unfrequently attention is first attracted by blocking of the nose, either from the swelling of the mucosa or from the presence of polypi. The symptoms are generally unilateral, but bilateral antral empyema is by no means rare. Pain and tenderness over the region of the antrum, or dental neuralgia, is occasionally noticed. The pain over the antrum may be sometimes described as boring, nibbling, or bursting. Infra-orbital neuralgia and tenderness over the infra-orbital foramen is fairly common; on the other hand, the pain may be supra-orbital with a sense of burning or boring over the corresponding frontal sinus.

Again, in other patients, severe headache, sometimes unilateral, is the most notable symptom, sometimes associated with mental depression, giddiness, insomnia or drowsiness, a dragging pain at the back of the eye; and indistinctness of vision on the side corresponding to the affected antrum, is a frequent symptom. The unilateral discharge of pus is especially liable to occur on rising from bed, or on stooping, or lying with the head on the sound side.

The patient usually complains of a constant or intermittent factor, and this *causmia* may be the only symptom noticed when the purulent secretion is small in quantity. In others, loss or perversion of taste is the prominent symptom.

When, owing to occlusion of the ostium and absence of any apertures of exit, the pus is permanently retained, the antral cavity may become distended. A smooth, hard, tender swelling then appears on the cheek corresponding to the antrum, although such distension of the cheek is more commonly due to either cystic disease or myxomatous polypi. The thin and partly membranous internal wall more usually yields first, and next in frequency, the floor of the orbit is pressed upwards with resulting displacement of the eye upwards, or, very rarely, forwards (exophthalmos). It is quite exceptional for a fistula to occur, but if it does form, it is almost always in the nasal wall, the weakest point.

Though permanent occlusion or absence of the *ostium* is comparatively rare, temporary occlusion is not unfrequent, the symptoms and physical signs becoming greatly aggravated, with some redness and swelling over the cheek, in the canine fossa, or on the outer wall of the nasal passage, till with increasing pressure the aperture is forced by the accumulated secretions, the recurrence of the nasal discharge being accompanied by relief of symptoms and subsidence of the localised swelling.

Various secondary symptoms may develop, and these may become so pronounced that their actual cause be overlooked, *e.g.*, defective vision, lachrymation, pains in the orbit, disturbances of taste or smell, paroxysmal sneezing: or, again, headache, sometimes frontal, sometimes parietal, occasionally occipital, may be the chief or only complaint. The pus escaping into the pharynx may set up catarrh of the Eustachian tube and consequently deafness and tinnitus; or by trickling down to the larynx produce violent attacks of coughing and choking on lying down at night. The pharynx also may be so irritated and inflamed that swallowing is painful.

Diagnosis.—The chief indications of antral empyema are:—

(1.) *Pus in the Middle Meatus.* The most constant sign is the presence of a small bead of pus appearing some way back beneath the middle turbinal. Pus in this spot may come from any of the anterior group of sinuses, but if after wiping the pus away it re-appears in the spot *immediately*, the evidence of antral empyema is strong, and the pus may sometimes be made to appear by gentle pressure over the antrum.

(2.) After tilting the patient's head laterally so that the suspected antrum is uppermost, I have often found that the pus may be watched welling into the middle meatus during anterior rhinoscopy.

(3.) *Fränkel's Sign*.—Pus reappears on the patient inverting his head and tilting it so that the sound side is uppermost. Inversion of the head does not cause a flow of pus from the frontal sinus, but occasionally pus in the anterior ethmoidal cells does. The amount of pus coming from these small anterior ethmoidal cells is always small and very seldom accumulates in sufficient quantity to flow out on inverting the head.

(4.) *Cacosmia, or Subjective Fœtor*.—The pus is generally fœtid, in which case the odour is perceived by the patient except when the antral disease is a complication of ozæna—a very rare association in my experience. This subjective fœtor, without obvious cause referred to one nostril only, is often a very important symptom.

(5.) *Pathological conditions in the Mucous Membrane*.—When no pus is to be seen in the nasal passage, we may still be led to suspect antral empyema from the secondary changes in the middle meatus. Most frequently œdematous polypi are found, or œdematous granulations round the margin of the *hiatus*, together with redness and swelling of the mucosa of the inferior meatus. But in place of swelling there may be a dry chronic catarrh of the nasal passage on the affected side.

The condition of the teeth will sometimes throw some light on the case. They may be all quite sound except those corresponding to the diseased antrum, and these may be carious or have "dropped out."

There are three other methods by which the diagnosis may be rendered more certain:—

(1.) By *transillumination*, as suggested by Voltolini, and developed by Heryng. A small, four-candle-power electric lamp is placed in the mouth, which is closed, the room having been rendered absolutely dark. In a normal patient the nasal passages and the cheeks become illuminated by a rosy-red



FIG. 177.

Anterior view of the nasal passages of a patient with empyema of the right antrum. The right middle turbinate appears to be double, owing to the swelling of the external wall of the middle meatus, but the pus is really escaping between the middle turbinate and the swollen mucous membrane referred to.

transmitted light, the patient often perceiving a sense of light himself. Any difference in the amount of light transmitted is

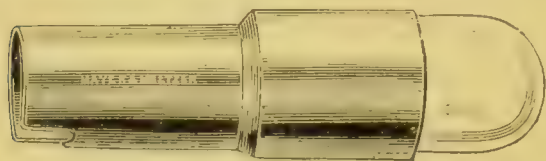


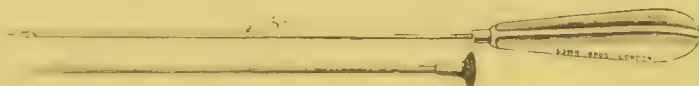
FIG. 177.

Heryng's Lamp for electric transillumination of the antrum.

estimated, and if pus be present in the antrum, or if it is the seat of a solid tumour, there is generally an obvious shadow on the affected side, more especially in

the infra-orbital region, and the patient himself will often perceive light on the sound side only.

Sometimes the pupil of the eye on the side of the empyema remains dark, instead of being illuminated like its fellow on the sound side. If the lids be kept closed during illumination, the patient often perceives the light on the sound side only, or more strongly than on the side of the empyema. Transillumination is often of great service, but cannot be absolutely relied upon for any inflammatory thickening of the mucous membrane of the antrum, or a difference in the thickening of the bony walls may produce an umbra on the affected side, while a cystic tumour may rather increase than decrease the brilliancy of the transmitted light.



FIGS. 179 and 180.

Lichtwitz' Trocar and Cannula.

(2.) *Exploratory puncture* by means of a Lichtwitz trocar and cannula, or with a strong exploratory hypodermic needle passed through the wall of the *inferior* meatus; or by drilling through the socket of a decayed tooth after its extraction. The middle meatus is readily entered by an exploratory trocar or needle, as the outer wall of the nose is thinnest here, but though this route has been advocated, it is less satisfactory than the former, and is, moreover, attended with a risk of the needle entering the orbital cavity. By whichever way the cavity is entered pus may well out or may be drawn out through the needle or trocar. But if it does not appear, the cavity should be washed out, or peroxide of hydrogen injected. If pus is present it is driven out and fills the nose as a white foam.

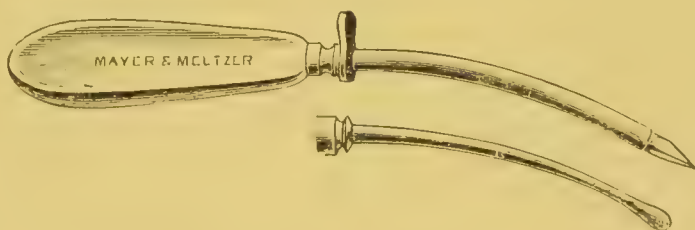
(3.) *Irrigation through the natural orifice.*—Having applied cocaine to the middle meatus, introduce a syringe with a curved nozzle through the ostium maxillare, and irrigate the antral cavity with a solution of peroxide of hydrogen (10–20 vol.). This method is excellent when it can be carried out, but in many cases it is a very difficult or impossible manipulation.

Treatment—The treatment of cases of antral disease requires much care and perseverance on the part of both patient and attendant. In all methods the object is the same, viz., to evacuate the pus and cleanse the antral mucous membrane with some antiseptic and stimulating application. If any polypi or hypertrophies of the mucous membrane obstruct the natural orifice, these must first be removed.

There are several alternative methods of procedure :—

(a.) A few rhinologists irrigate through the natural opening. A special syringe with a curved nozzle or a Hartmann's cannula is directed through the ostium and the solutions injected. If treatment is attempted through this orifice it should be enlarged, and the anterior portion of the middle turbinal may have to be removed so as to allow freer access. Good results have been obtained by daily irrigation of boric acid solution through the natural opening into the antrum.

(b.) By irrigation through an artificial orifice in the outer wall in the inferior meatus. This method of Krause and Mickulicz has its disadvantages, since it is difficult to secure good drainage, the floor of the antrum being well below the floor of the nasal passage. Moreover, the orifice is necessarily too small to allow curetting.



FIGS. 181 and 182.
Krause's Trocar and Cannula; and Guide.

Krause's curved trocar and cannula is probably the best instrument to use for perforating the wall of the meatus, after the application of cocaine. The trocar should be made to enter the antral cavity just below the nasal duct and close to the floor

where the wall is thin, care being observed to prevent its suddenly impinging on and injuring the outer wall of the antrum. Then withdrawing the trocar, with the cannula in the antrum, the cavity is washed out thoroughly with an antiseptic solution. The operation is often attended with considerable hæmorrhage. Finally, some antiseptic powder is insufflated. This must be repeated daily for a time by the physician, and then, as the pus secreted diminishes in quantity, less frequently till all trace of purulent secretion disappears. With a small cannula having a curved lip the patient can soon learn to find the opening so as to wash out the cavity himself.

Walter Freeman has improved on the method of Krause and Mickulicz by entering the antrum more anteriorly.

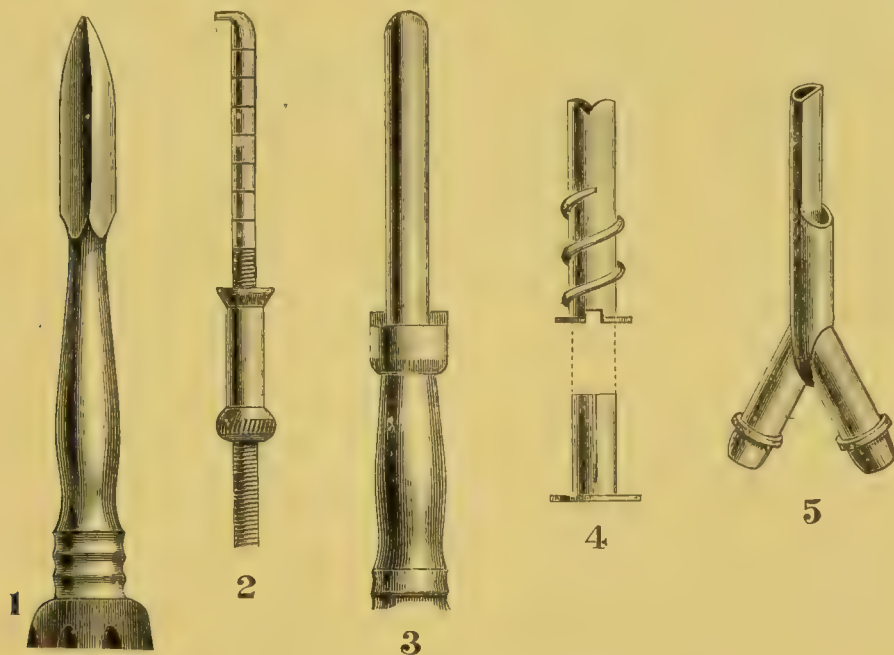
(c.) By an opening into the antrum through the alveolus, a method adopted by Cowper as far back as 1698. In many cases a carious upper molar is present, or the upper teeth corresponding to the affected antrum have been lost. The best point to drill through to enter the antrum is an outer socket of the first molar, but the second bicuspid or second molar socket may be entered if it is desirable to retain the first molar either because it is a sound tooth or because it is necessary for mastication.

The patient being anesthetised by nitrous-oxide, the tooth or stump is extracted and the drill firmly held in the hand is made to enter the antrum through the outer border of the alveolus, being directed upwards and slightly inwards. If directed too much inwards or outwards, the narrow floor of the antrum may be missed, and, in the former event, the floor of the nose punctured instead. The operation is usually simple and free from risk, though one case of alarming hæmorrhage, possibly due to an angiomatous condition of the lining mucosa, is reported by Scheppergrell.

The pus may well out of the opening thus made, but it is often necessary to wash out the antrum by injecting some warm water, the fetid secretion mixed with water running out of the nose. A still better plan is to inject a *few drops* of hydrogen peroxide, which, if it encounters pus in the antrum, at once causes a rush of foam in the middle meatus, and often at the newly-made alveolar opening. The quantity of hydrogen peroxide injected for diagnostic purposes should be small, because by throwing in enough to fill and overflow the antrum into the nose it would at once foam up in the presence of pus in the nose itself, and its value for diagnostic purposes lost.

The diagnosis having been corroborated by the finding of pus, the antrum is washed out thoroughly with some antiseptic lotion, and the peg inserted.

In my experience the best method of entering the antrum through the alveolus is by means of the ingenious instruments devised by my colleague, Mr. W. R. Ackland. They comprise : (1) A borer of special size, which readily cuts the bone of the alveolus when rotated forwards (like a screw) ; (3) A tube carrier, on which the antral tube fits ; (4) A silver-gilt antral tube, having a screw thread outside which retains it when *in situ*, and a "split-pin" stopper ; and, (5) A two-way nozzle, which fits i to the tube when it is desired to wash out the antrum. A single-way straight tube may be used instead, flushing through the nasal opening. Further, a measurer (2) is provided for use when it is desired to measure the depth of the bone traversed before the antral floor is reached.



FIGS. 183 to 187.

Ackland's Antral Empyema Instruments. For description see text.

The patient being under nitrous-oxide anæsthesia, the tooth is extracted (when necessary), and the antrum bored. The tube on the carrier is immediately screwed into position, and the operation is concluded on the withdrawal of the carrier. The antrum is then washed out by means of the two-way nozzle, and finally the stopper is inserted to close the lower end of the tube in the antrum. By injecting the fluid rapidly into the antrum a good flush through into the nasal passage is obtained.

Various siphons and douches have been devised for the daily lavage of the antrum through the alveolar opening ; a simple method is to use a small syringe with a curved vulcanite nozzle.

The advantages of this method are that the patient can syringe out the antrum himself ; the operation causes very little after pain or disturbance, and the drainage is from the lowest point in the floor of the antrum.

Thorough syringing must be carried out, at first twice daily, then as the amount of discharge diminishes, once a day, until the secretions have been free from pus and odour for at least a fortnight. In this way I have sometimes obtained a cure in a few weeks; but in other cases, the process of syringing the cavity daily may have to be continued for months before the unhealthy discharge disappears. The antiseptic solutions should be warmed before injection. I find it is a good plan to use a strong solution of peroxide of hydrogen (20-30 volume) to thoroughly cleanse the cavity first; it ferrets out every socket of curdy pus in a very short time. When the cavity has been well cleared, boracic acid, biniodide of mercury (1 in 2,000), iodine water, permanganate of potash, chloride of zinc (grs. 1 or 2 to the ounce), or common salt solution, may be injected until the lotion returns clear and free from odour.

(d.) Entry through the canine fossa.

The upper lip being well drawn up, an incision is made in the gingivolabial angle, right down to the bone, extending horizontally from a point corresponding to the canine tooth, backwards as far as the first molar; then the periosteum and mucous membrane having been reflected, a hole about the size of a sixpence is trephined into the antrum in the canine fossa, passing directly backwards with a slightly upward direction, or the opening may be made with a chisel or mallet, the bone being chipped away down to the level of the antral floor.



FIG. 188.

To show the place where the superior maxilla should be trephined for antral disease.

The antral cavity should be thoroughly washed out with warm water, and when all pus and inspissated secretions have been removed, the interior of the cavity should be inspected, or the finger inserted to explore for the presence of polypi or papilliform thickening, which should be freely curetted. Any

bony septa that may interfere with drainage, should be broken down. Finally, the cavity should be cleansed and packed with iodoform

gauze. This should be removed, and the cavity washed out daily with some antiseptic, or if syringing be alone relied on, it should be carried out three times daily at first, and then twice a day till the discharge has completely ceased. After a time we may use some stronger and more stimulating injection, such as chloride of zinc (grs. v ad ʒi), or a weak aqueous solution of iodine, and the cavity gradually allowed to close.

(c.) The method introduced independently by Caldwell and Scanes Spicer is a combination of the intra-nasal method with the canine fossa route. The opening into the antrum is made as in the preceding method. The inner surface of the antrum is then curetted so as to remove any granulation tissue, etc., after which, with the finger introduced into the cavity to act as guard, Krause's trocar and cannula are passed down the inferior meatus of the same side, and with it a large opening into the antrum is made well behind the nasal duct (see *Frontispiece*). The special feature in this method is the immediate closure of the canine fossa opening, so that all the washing out and drainage is subsequently made through the opening in the inferior meatus.

Luc, in 1897, described a similar method which had proved very successful. After curettement guided by electric illumination, he swabs out the cavity with strong chloride of zinc solution, and then sews up the opening in the mucous membrane with catgut, and in one case it united by first intention at the end of three days.

Scanes Spicer irrigates the antrum and nose with warm boracic lotion, and the cavity of the antrum is then tightly packed with creolin gauze for forty-eight hours. At the end of forty-eight hours the gauze is removed and no drain is used at all, but free irrigation made three times a day, and the patient instructed to blow air through the antrum, first by the nose and then by the mouth, and to use boracic lotion frequently in the same way. By this means the antrum is more effectually cleansed, and a source of irritation in the drainage tube is dispensed with.

What are the indications for each method? — Individual opinions differ widely on this point, but speaking generally the method of opening through the alveolus should be first tried as a matter of routine, because drainage is from the lowest point in the floor, and the patient can carry out lavage of the cavity himself, while the operation is readily done under nitrous oxide gas without being followed by any notable pain. In a certain number of cases this simple method of alveolar drainage fails,

and the suppuration continues for a long period; the chief causes of failure being either (1, the extensive formation of polypi and papilliform thickenings of the mucous membrane of the cavity, or (2,) the co-existence of purulent inflammation in the ethmoidal or frontal cavities, from which pus escapes into the corresponding maxillary antrum. It is then better to resort to the more radical method of opening through the canine fossa, so as to permit of thorough curetting. Even in very chronic cases a good result may often be obtained by the alveolar opening. Probably granulations and papilliform thickening of the mucous membrane are present in nearly all chronic cases, but these are caused by the retained decomposing pus, and with the removal of the cause the mucous membrane returns to a healthier condition.

When the alveolar method has failed, the Caldwell-Spicer method affords a rapid and almost certain means of obtaining a permanent cure. By closing the opening in the canine fossa at once, much discomfort and inconvenience is obviated, and the patient will very quickly learn to wash out the cavity through the nasal opening in the inferior meatus by means of a douche with a fine, curved nozzle—an ordinary Eustachian catheter answers admirably. Nevertheless, the cavity has sometimes to be re-opened and curetted afresh, and though such a contingency is very exceptional if the counter opening has been made in the nose so as to permit of lavage for a long period if necessary, it is not so rare if the opening in the canine fossa alone is relied on.

Various circumstances may make a departure from the customary methods of procedure desirable: for instance, the patient may have no useful teeth on the sound side, and may have to rely on a tooth which would have to be sacrificed if the alveolar method was employed, or the tooth may be required to retain his dental plate. Again, a patient may have only a few weeks to spare for treatment, and prefer the most rapid and certain, yet more severe method.

CHRONIC FRONTAL SINUSITIS.

Symptoms. Empyemas of the frontal sinus may be divided into three groups: (*a*,) Closed; (*b*,) Open; and, (*c*,) Intermittent or alternating.

A closed empyema, or, if it be a catarrhal inflammation without suppuration, a mucocoele, produces external signs if there

is yielding of the walls, and as the inner part of the floor corresponding to the inner angle of the orbit is the thinnest wall, this generally is the usual seat of the external swelling. The eye is displaced downwards and outwards, and since this and the orbital swelling are the most notable features presented, these rare cases are usually referred to ophthalmic surgeons.

On the other hand, the anterior wall or the posterior wall may yield, in the one case producing a facial deformity, and in the other symptoms of compression of the frontal lobe of the cerebrum.

But usually the earliest symptoms are central frontal headache or supra-orbital neuralgia.

The headache is sometimes intermittent, increased by exertion or by pressure, mental effort, or the use of alcohol, and very often resembling migraine, being attended with nausea and vomiting, or with an intermittent feeling of falling. With increasing secretion the headache is generally severe and almost unendurable till, with the escape of yellow pus or muco-purulent secretion, the symptoms are mitigated.

Even in "open" frontal empyema, which is much more common than the "closed," frontal or super-orbital headache is usually a marked symptom; sometimes the headache is occipital. In other cases again the headache is not severe, hardly amounting to more than a dull, heavy feeling over the frontal sinus, aggravated by stooping and especially by pressure over the upper internal angle of the orbit. Loss of appetite, diurnal somnolence, and general mental dulness and apathy may be present. But often enough the symptoms are of a very indefinite character in the absence of complications. When the frontal nasal duct is not closed there is discharge of pus from one nostril only, unless both frontal sinuses are involved.

When the pus escapes into the nose, it will be found in the middle meatus on anterior rhinoscopic examination, and after cleansing the passage and wiping away the pus with a pledget of wool it usually re-appears after a short interval, towards the anterior extremity of the middle turbinal. If the pus be small in amount, Grünwald suggests the plan of keeping a pledget of cotton-wool tightly packed in the middle meatus to temporarily dam up the secretion, so that it flows again more freely on removing the pledget. Pus here must come from one of

the anterior group, but in frontal sinusitis it does not re-appear more freely after being wiped away by lowering the head, as in maxillary empyema. If by this and other signs maxillary sinusitis is excluded, we have then to consider only frontal sinus and anterior ethmoid cell disease, which very often co-exist. If on probing the bulla ethmoidalis and the region above it, no softening or bare bone is detected, it is probable that the lower cells of the anterior ethmoid group are not the source of the pus. In frontal empyema the pus escapes more readily in the upright position, and so the patient finds it most abundant on rising in the morning, continuing more or less persistently throughout the day. It is not generally very foetid, but the patient often complains of the smell, *cacosmia*.

Complications. (a.) Firstly, there are those in the nasal accessory sinuses due to implication of the anterior ethmoidal cells and the corresponding maxillary antrum. The association of purulent sinusitis in several of the accessory nasal cavities has already been discussed, but it may here be remarked there are relatively few empyemas of the frontal sinuses alone, the majority of cases being complicated by implication of the ethmoidal cells with all their attendant dangers.

(b.) Secondly, the continued flow of pus causes inflammatory changes in the nasal mucosa. Polypi are very often found crowding round the lower end of the infundibulum, and atrophic changes resembling *ozæna* may arise.

(c.) Thirdly, apart from the displacement of the eye in closed frontal sinus, dacryocystitis, redness and tenderness in the inner angle of the orbit and aching in the eyeball are met with in latent cases. Again, if congenital lacunæ are present in the inner part of the floor, or from softening of the wall perforation takes place towards the orbital cavity, suppurative cellulitis will arise, phlegmon of the orbit being most frequently due to this cause according to German.

Lastly, cerebral complications may occur, either due to pressure from yielding of the posterior wall compressing the frontal lobe, or from perforation following erosion of the wall, with resulting suppurative meningitis or frontal abscess. But an abscess in the frontal lobe may be produced without any bone lesion, micro-organisms being carried directly by the lymph channels or blood-vessels, just as a temporo-sphenoidal abscess

may arise in suppurative otitis, and in a few cases the posterior wall of the sinus may be congenitally defective.

Another fortunately rare complication is the occurrence of septic osteomyelitis involving the diploic sinuses, tending to become diffused over the whole cranial vault. Instances of this fatal complication have been recorded by Luc and Tilley.

Diagnosis. Closed frontal empyema generally presents such definite external features as to leave no difficulty in the diagnosis, but the external deformity may be simulated by various tumours, or by a dermoid cyst.

It is, however, often very difficult to diagnose a latent frontal empyema with certainty, and one has generally to rely largely on the exclusion of other sources for the pus appearing. Many cases simulate migraine, and as the amount of pus escaping may be insufficient to attract the attention of the patient, it is well to remember the possibility of frontal sinus disease in patients suffering from severe intermittent headache which does not yield to treatment.

Electric transillumination, by the method of Vohsen, occasionally assists diagnosis by excluding frontal sinusitis in some cases. The patient being in an absolutely dark room, a small electric lamp is placed as deeply as possible under the floor of the sinus,

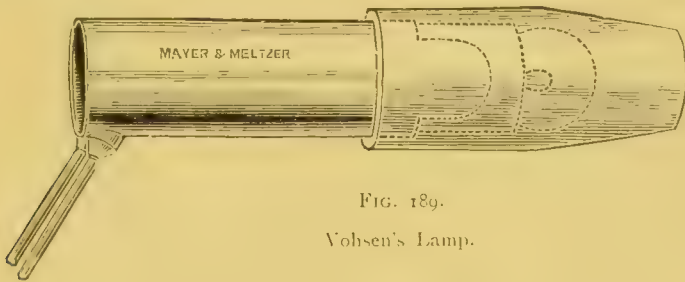


FIG. 189.
Vohsen's Lamp.

in the upper internal angle of the orbit, first on one side then on the other. In the normal subject, the region of the frontal sinus is often lit up by a diffuse pink light extending a little beyond the median line, but when the frontal sinus is full of pus, this region remains dark. If the suspected frontal sinus lights up well, it affords some evidence against the presence of pus, but if, on the other hand, the suspected sinus remains darker than the other side, the sign is of little value, inasmuch as the anterior walls are very often of unequal thickness.

Logan Turner, from the examination of five hundred skulls, has shown how little reliance can be placed on transillumination of the frontal sinus. Thus, in 357 specimens with both sinuses present, 98, or 27 per cent., failed to illuminate on both sides; 50, or 14 per cent., failed on one side; 40 illuminated on the opposite side, and 23 were negative on the opposite side. When the frontal sinuses were absent, the frontal area never illuminated.

The passing of a probe a *short distance* up the fronto-nasal duct can be effected in some cases without using any force, and is then permissible. If pus is present it will often well out alongside the probe at the lower aperture of the infundibulum. A probe from $\frac{1}{2}$ to 2 millimètres thick is bent at an angle

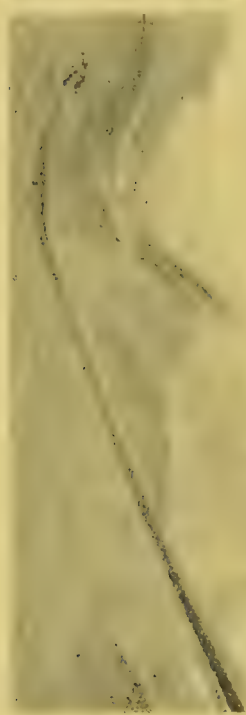


FIG. 190.

Skiagram showing a bent probe passed into the Right Frontal Sinus $\frac{1}{2}$ size. The extreme top of the probe was ill-defined, and has been dotted in.

towards the point. I usually bend a probe so as to form a quarter circle with the terminal 30 millimètres. Hansberg bends the last 30 millimètres of the probe at an angle of 120° , and Lichtwitz bends the probe at a right angle 10 millimètres from the end. The probe is passed up into the angle between the outer wall of the middle meatus and the middle turbinal, and carried forwards until the point is felt to enter the infundibulum, when it is passed directly upwards for about 1 cm. and then up slightly forwards, so as to avoid entering the ostium of one of the anterior ethmoid cells, for another 1 or 2 cm. Further than this I think it is not altogether safe to go, for reasons stated further on, but some rhinologists do not hesitate to pass the probe up for 5 or 6 millimètres when it may be considered to have certainly entered the sinus. In a large

percentage of cases it is impossible to make a probe enter the infundibulum even when the anterior portion of the middle turbinal has been first resected. This partial turbinectomy should always be performed if it is desired to pass a probe or irrigate the nasal passage, unless, as rarely happens, the opening of the fronto-nasal duct is soon found. The method of passing a probe into the frontal sinus under the guidance of the X-rays, as carried

out by Spiess, renders the procedure much less dangerous, as the operator can watch the point of the probe in its upward passage, and see when it has reached and entered the sinus.

Exploratory lavage of the frontal sinus through the natural orifice has been advocated, and in a very few cases it is not particularly difficult. But even when it is possible, the information afforded is not very precise, inasmuch as it is very difficult to

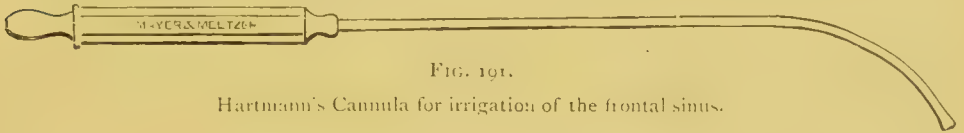


FIG. 191.

Hartmann's Cannula for irrigation of the frontal sinus.

decide whether the pus washed out is from the frontal sinus or from the upper anterior ethmoidal cells. In making exploratory lavage a specially-constructed cannula with a fine bent nozzle is passed up between the outer wall of the middle meatus and the middle turbinal in the manner adopted in passing a probe.

Mink has devised an ingenious arrangement by which he can apply one end of a tube over the frontal sinus, the other end being inserted in the external auditory meatus of the physician. Then a fine catheter attached by a rubber tube to a rubber ball is inserted by its free end into the lower end of the infundibulum. By blowing air into the sinus he can detect bubbling if empyema exists, just as we detect fluid in the middle ear.

Treatment.—As a preliminary measure, any polypi or granulations in the middle meatus should be removed, and the part thoroughly curetted, and any œdematous or hypertrophic mucous membrane around the hiatus semilunaris bound down by the galvano-cautery. Further, to gain access to the lower end of the infundibulum and to remove any possible obstruction here to free drainage, the anterior portion of the middle turbinated body—the operculum—should be removed by Grünwald's method, as follows: either by the application of cocaine or under nitrous-oxide gas, the anterior third of the turbinated body is divided by bent scissors close to its attachment to the outer wall of the nose (for this purpose Charters Symonds' scissors are very convenient), and then separated by a strong wire snare. The treatment of the frontal sinus may then be undertaken by various methods which may be divided into the intra-nasal and external methods.

The intra-nasal method consists in removing all secretion from the nasal passages, and by means of a syringe bearing a specially-constructed cannula, as in exploratory lavage, frequent irrigation of the frontal sinus with warm mild antiseptic lotions through the infundibulum. The treatment consists essentially in providing effective drainage for the purulent secretions. The large majority of acute and probably a considerable number of cases of chronic purulent frontal sinusitis recover spontaneously, the natural orifice in the floor being in the most dependent part of the cavity. The cases that come under the care of the rhinologist are usually those in which there is obstruction to the free escape of the pus from various causes *e.g.*, an unusually narrow fronto-nasal channel generally due to encroachment of the ethmoidal cells, or narrowing of the nasal duct from pathological changes in the lining mucous membrane), and therefore are not as a rule likely to permit of successful irrigation. The passage of a trocar or curette into the frontal sinus is a dangerous procedure, because even in healthy subjects, the thin cribriform plate may be so easily perforated, and in some individuals the posterior wall of the frontal sinus is absent, or congenitally defective so that there is no bony wall at all to prevent the instrument entering the cranial cavity.

We may recall Mermod's case in which meningo-encephalitis followed exploration of a supposed frontal sinus by a probe. It was proved *post mortem* that the frontal sinuses did not exist, and the liquid that had escaped by the nose during life could only have been cerebro-spinal fluid. There were two holes in the base of the anterior fossa, just behind the usual situation of the frontal sinuses.

We must remember, too, that the thin walls are often much softened or eroded in chronic empyema, and the passage of a trocar or probe, however carefully done, might readily lead to complete perforation into the anterior fossa of the skull, and in a less degree the same objections apply to irrigation unless they be most cautiously and skilfully carried out.

Notwithstanding the drawbacks to the intra-nasal methods they may sometimes be adopted with complete success. Chiari, for instance, has recorded the cure of two cases by these means. Much depends on the anatomical construction of the parts, which we have seen is very variable in different individuals; for

instance, Lothrop has pointed out that in more than 50 per cent. of the specimens he examined the fronto-nasal duct did not exist, the ostium frontal opening almost directly into the middle meatus by means of little or no canal. Such sinuses are sometimes comparatively easy to probe and to irrigate, especially if entry into the frontal sinus was guided and directed by the X-ray screen, as carried out by Spiess, and in latent empyema it would be not only quite justifiable but desirable to give the intra-nasal method a trial before resorting to more radical external procedures, which with many advantages have certain disadvantages too, apart from the risk, always present, of leaving an ugly cicatrix. Curettement of the sinus through the nose, however, is a most dangerous procedure, though it has been successfully carried out. Curettement of the *lower portion* of the passage between the sinus and the nose is less open to objection, and may greatly facilitate the irrigation.

A further objection to the intra-nasal method lies in the practical inability to attack by this route the very frequently associated ethmoidal-cell empyema. Zuckerkandl has stated that he has never met with a single suppurative case of isolated inflammation of the mucous membrane of the frontal sinus, and Bryan has drawn attention to the frequency with which the fronto-ethmoidal cells are involved in either frontal sinus or ethmoidal cell suppuration; and "if affected, as they generally are, they will be starting-points for a re-infection of the sinus after the inflammation has apparently subsided."

External operation.—There are several external methods of gaining access to the frontal sinus; mostly differing in detail only, but these methods fall into two divisions: (*a*.) Entry through the inferior surface; and, (*b*.) Entry through the anterior surface:—

(*a*.) *Entry through the inferior surface.*—An incision is made commencing from a point on the nasal process of the inferior maxilla, in front of the margin of the orbit at the level of the inner canthus and extending upwards and slightly outwards to meet the eyebrow, and then along the lower margin of the shaved eyebrow as far as the supra-orbital notch, the angular and supra-orbital arteries being divided. The periosteum and soft tissues are reflected downwards so as to expose the orbital surface or the floor of the sinus, and with a small trephine or chisel and bone forceps an opening is made in the thin bony floor just above the internal angular process of the frontal bone. The lining

membrane is then divided, and the sinus explored with a probe. A probe is passed down the fronto-nasal duct to the nose to serve as a guide in further enlarging the opening and removing the inner portion of the floor of the sinus. The anterior ethmoidal cells should be exposed, and, if they are involved, should be broken down by small curettes in a direction downwards, backwards and inwards, so as to avoid injuring the cribriform plate above, or the *lamina papyracea* externally.

(b,) *Entry through the anterior surface* is the operation devised by Ogston in 1884, and independently revived by Luc in 1896, is now known as the Ogston-Luc operation. In performing the Ogston-Luc operation, the inner half of the eyebrow having been shaved, a curved incision is made along the inner third of the superior orbital margin, and carried down to the bone along the corresponding margin of the nose to the naso-frontal suture, and the periosteum having been reflected, the anterior wall of the sinus is perforated with a small trephine. The opening then made is enlarged with cutting forceps to the size of a sixpence, or a two-fifths of an inch or larger trephine may be used to remove the bone, applied just above the supra-orbital ridge and internal to the vertical line corresponding to the inner canthus. The interior of the sinus is then exposed by incising the lining mucosa, examined, and explored in every direction with a probe, and the diseased, thickened mucous membrane freely curetted. To enable one to do this, it is usually necessary to enlarge the opening with bone forceps, but if the sinus does not extend laterally more than a few millimètres outside the trephine opening, such extension of the opening may be unnecessary. The subsequent steps in the operation apply equally whether the entry be made through the floor or the anterior wall. The septum should be explored to ascertain if there is any communication with the other sinus. If the two sinuses communicate, or if there is evidence of both sinuses being the seat of empyema, the septum should be completely broken down and the whole operation done on both sides. It is not safe to rely on both cavities being drained through one nasal duct. The probe should be passed into the nose through the naso-frontal duct, and then, with this as a guide, the posterior nares and rhino-pharynx having been tamponed to prevent blood and pus running back into the larynx, a trocar should be passed down to the nose, or the duct may be freely curetted. Any bony obstruction here ought to be broken down so as to ensure a free passage for future drainage into the nose. The floor should be attacked far back, as it is thinner here than in front. The ostium lies deeper than is generally believed; Tilley found that it may be as much as 28 millimètres from the anterior surface. At this stage, if not already done, the condition of the frontal-sinus and frontal-ethmoidal cells should be explored by the probe, and if softened or affording any indication of inflammatory disease, these too should be freely but cautiously curetted and opened with a small, sharp spoon, or other instrument, in a downward direction, so that their free communication with the enlarged fronto-nasal canal is ensured. Some operators make a very free communication

with the nose by using a sharp spoon or curette ; others, finding that however large the opening it tends to cicatrise and contract, do not use the curette at all.

The whole cavity and enlarged duct should be freely swabbed out with chloride of zinc solution (grs. xx to xxx ad ̄5j). Two alternative methods of completing the operation are available : (a,) Either the whole cavity may be packed with a thin strip of antiseptic gauze, and the external wound closed except at its inner angle, which is left with one end of the gauze strip projecting. A strip of gauze, too, should previously be packed into the naso-frontal canal, the lower end projecting into the nose to facilitate removal ; (b,) Or, if preferred, a Luc's rubber drainage tube with an upper enlarged open end (to retain the tube when *in situ*) and a lower rounded enclosed end, is carried on a bent probe through the fronto-nasal passage from above till the upper end rests in the frontal sinus ostium, the lower end being drawn out at the anterior nasal orifice. The probe is withdrawn, the part of the drainage tube projecting from the nose cut off, the periosteum drawn down and sutured, and the wound closed and dressed. The presence of a drainage tube sometimes tends to excite the formation of granulations, which on removal of the tube cause stenosis and cicatrization.

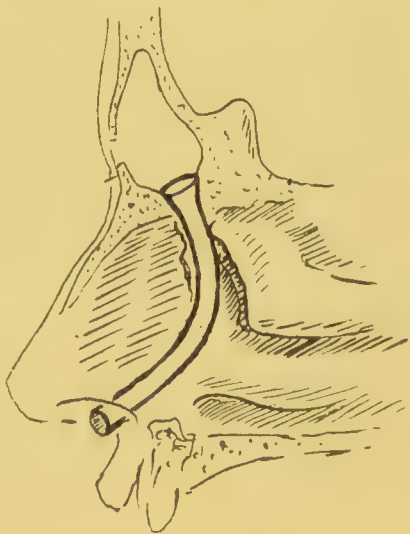


FIG. 192.

Diagram showing a Rubber Drainage Tube, for draining the frontal sinus, in position.

The sinus should be washed out daily through the drainage tube with warm boracic acid, normal saline, or very weak biniodide of mercury solution, and it is a good plan to pass a small flexible tube (Weber-Liel or intra-tympanic tube) up through the lumen of the drainage tube, through which the antiseptic fluid may be injected, as recommended by Waggett, as "by this means free exit is afforded for escape of the lotion, and danger of extravasation into the soft tissues of the forehead avoided."

As the secretion diminishes in amount, the irrigations may be made at longer intervals, till the contents of the sinus have remained free for a week, when the drainage tube is withdrawn through the nose, and the case is cured.

When the tube is dispensed with and the wound has been packed with gauze, the gauze should be removed piecemeal, a few inches being cut off daily when the wound is dressed, so as to allow the whole cavity to granulate up from the bottom. In a female it is better to employ the drainage tube with immediate closure of the external wound, and run the risk, in case of failure to obtain a cure, of having to re-open the

cavity with recourse to the gauze and packing method, which is a little more likely to leave a slightly puckered cicatrix at the inner angle.

The operation often leaves no deformity at all, especially when the anterior wall has not had to be removed to any great extent. Ogston made a median incision from the root of the nose upwards, and many operators still prefer this to the supra-orbital incision, the advantage urged and so ably advocated by Mayo Collier being that a large trephine opening in the middle line enables one to explore both sinuses, yet though in males the central cicatrix remaining is not a great deformity, in females it would be considered unsightly, and moreover if both sinuses are involved there is no reason why the Ogston-Luc operation should not be performed on both sides.

There are numerous modifications of the methods described, as regards the incisions, the amount of the wall removed, the treatment of the fronto-nasal passage, etc. Thus some advise complete removal of the anterior wall, aiming at complete obliteration of the sinus; Jansen seeks to attain this end by removing the inferior surface of the sinus and making a large opening into the nasal fossa, while Kuhnt removes both the anterior and inferior walls, and Röpke has obtained very rapidly successful results by this operation. The substitution of a silver plate beneath the integuments for the removed anterior wall has been successfully used to prevent any depression and deformity.

For most cases entry through the floor of the sinus is preferable, inasmuch as one obtains more complete access to the fronto-ethmoidal cells, though the sinus itself is less completely exposed. Especially is this route to be preferred when a fistula has already formed. The lower route has the disadvantage, however, of increasing the liability to orbital inflammation, and if due care be not observed the pulley of the superior oblique muscle may be permanently displaced, causing squint.

Some operators still adhere to older methods of draining the sinus through the external incision, but unless a sinus has already formed, it is undesirable, as the orifice left by the drainage tube often creates an intractable fistula, which, even when it has been got to close, leaves an unsightly puckering. Yet it is sometimes better to adopt the more radical methods, and the plan of keeping the external opening unclosed for a time when such serious complications as erosion of the posterior wall has arisen, with or without meningo-meningitis or frontal abscess.

It is scarcely necessary to add that if antral empyema co-exists with the frontal sinus or ethmoidal cell suppuration, it should be dealt with at the same time by appropriate methods.

ETHMOIDAL CELL DISEASE.

The division of the ethmoidal cells into two groups, viz., the anterior and posterior, has been described already, and we have seen (*vide Fig. 176*, p. 362), that the anterior group are associated clinically with antral and frontal sinus disease, while the posterior

group are similarly associated with the sphenoidal sinus. The anterior group may further be sub-divided into:—

(1,) The *inferior cells*, including the bulla ethmoidalis, and cells opening immediately above it, and any cells in the middle spongy bone. The cells of this group are liable to become involved in inflammatory disease of the antrum, and cause polypoid degeneration in the middle meatus. They are not very difficult to reach, and may be opened up by intra-nasal methods without much difficulty.

(2,) The *middle cells*, just above the former, and lying to the inner side of the os planum, just below the level of the anterior ethmoidal canal. Great care is necessary in the operative treatment of these cells.

(3,) The *fronto-ethmoidal cells*, one or more forming the frontal bulla, when present. They lie around the ostium frontale below the floor of the nasal portion of the frontal sinus, some behind the fronto-nasal duct, another in front and corresponding to the lachrymal bone; but all generally open into the infundibulum or fronto-nasal duct. These are the cells which are particularly prone to participate in frontal sinus empyema.

ANTERIOR ETHMOIDAL CELLS.

Symptoms.—The ethmoidal cells are very frequently the seat of inflammatory disease, catarrhal or purulent, probably more often than any of the other accessory cavities of the nose. Mucous polypi in the middle meatus are by no means rarely due to ethmoidal cell disease without involvement of the other sinuses. Again, the anterior cells are also very frequently implicated in association with antral or frontal sinus empyema, and the posterior cells in association with sphenoidal sinus disease. Closure of the ostia, however, is rare, and thus the large quantities of muco-pus and the more definite or obvious symptoms

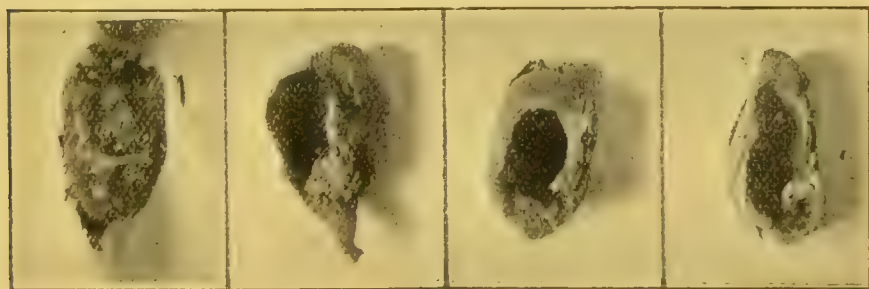


FIG. 193.

Examples of anterior Ethmoidal Cells at the anterior extremities of middle turbinals, showing formation of large bony cyst (WALKER DOWNSIE).

due to concomitant suppuration in the larger cavities often obscures the ethmoid cell affections, and results in their being overlooked.

The usual symptoms of inflammatory disease of the anterior cells are pain and tenderness on pressure at the root of the nose, the pain often extending outwards along the infra-orbital ridge to the temporal region. A deep-seated stupefying headache, torpor, dragging or aching at the back of the orbit may be present. Sometimes the irritation of the nasal mucosa causes symptoms resembling hay fever, sneezing and lachrymation.

When the cells become distended from retention of the secretions, the weakest wall yields, generally the lamina papyracea, and the eyeball is displaced outwards, diplopia resulting. Photophobia, lachrymation, and narrowing of the visual fields have been noted. A swelling forms on the inner corner of the orbit, often reddened and œdematous, and on pressure of this swelling the thinned bony wall may generally be felt to crepitate, and the pus is made to escape more freely into the nose. With the later involvement of the deeper layers of the mucous membrane erosion of the cell walls may occur, the pus becomes thicker, mixed with blood and sometimes bony particles, while epistaxis is not unusual. Though the pus is not usually very foetid, the patient complains of the smell.

Examination of the nasal passages generally reveals pus in the middle meatus, which reappears slowly after cleansing of the parts with a cotton-wool pledget. Mucous polypi or œdematous granulations are commonly present, and after these are removed pus will often well out from the ostia. The middle turbinal itself may contain the suppurating cells, and it is then red and œdematous, and the bone softened; or the ethmoid bulla may be enlarged, distended, and covered with inflamed œdematous mucous membrane, and may fill up the anterior portion of the middle meatus, descending lower than the middle turbinal, for which it may be readily mistaken.

A probe introduced cautiously beneath the middle turbinal may reveal softened carious bone and bony spicules of formative osteitis.

By electric transillumination with a lamp in a normal subject an illuminated area will sometimes show on either side of the nose, and, according to Ruault and Robertson, ethmoidal cell disease is revealed by absence of the luminous spot on the affected side. The sign is of doubtful import.

THE POSTERIOR ETHMOIDAL CELLS.

The posterior ethmoidal cells are rarely the seat of isolated inflammatory disease, but the condition is often very difficult indeed to diagnose, and many cases are probably overlooked. A subjective sense of thickness over the right frontal region, loss of memory, deep-seated stupefying dull headache, a sense of inability to concentrate the attention or to think clearly, aching of the back of the eye and obscurity of vision, associated with discharge of pus into the nose anteriorly and into the pharynx, seen on examination to occupy the olfactory fissure, and (by posterior rhinoscopy, see *Plate XXIV, Figs. 2 and 3*) issuing above the posterior end of the middle turbinal, constituted the symptoms in a case of mine, my diagnosis being corroborated by Sir Felix Semon. The absence of sphenoidal sinus disease was proved by passing a cannula into the sphenoidal sinuses and injecting hydrogen peroxide with negative results. This patient was wan, nervous and wretched, and his symptoms were always worse in damp weather, in the summer, and on blowing his nose.

When the cells become distended from occlusion of the ostia, the eyeball is pushed forward, or forwards and outwards, and suppurative orbital cellulitis, optic neuritis, etc., may occur.

Complications.—Apart from the complications resulting from extension of the diseased process to other accessory sinuses, suppuration in the ethmoid cells is liable to cause :

(*a*.) By extension upwards, through the cribriform plate, meningo-meningitis, abscess in the anterior fossa of the skull, or in the frontal lobe of the cerebrum ;

(*b*.) By extension towards the orbit, suppurative orbital cellulitis, and from involvement of the lachrymal duct, dacryocystitis, epiphora, etc. ;

(*c*.) Purulent inflammation in the lachrymal duct, etc., may also arise from infection by the nasal orifice ;

(*d*.) Eustachian catarrh, otitis media, catarrhal or purulent, with all its further possibilities are liable to occur, especially in disease of the posterior ethmoidal cells.

Fortunately the inflammatory disease is often limited to the lower cells of the anterior group, and many of the more serious complications are less prone to arise; nevertheless the extreme

difficulty of deciding the extent of the disease, and especially of dealing effectually with the condition when the upper cells are involved, renders it necessary to regard every case as a serious menace to life, and requiring careful and thorough treatment.

Treatment.—Mucocoele of the lower anterior ethmoidal cells usually recovers rapidly after opening up the involved cavities, but suppuration in the cells is less amenable to treatment.

In all cases the first step is to remove any polypi and granulation tissue, and to cauterise oedematous mucous membrane in the middle meatus.

When the anterior group is the seat of the disease, the anterior half of the middle turbinal should be removed by an incision

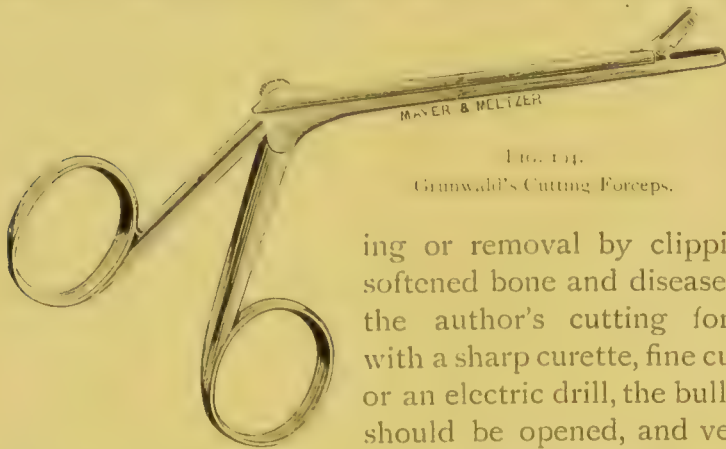


FIG. 114.
Grunwald's Cutting Forceps.

with scissors as close to its origin as possible, and subsequent snar-

ing or removal by clipping away the softened bone and diseased tissues with the author's cutting forceps. Then with a sharp curette, fine cutting forceps, or an electric drill, the bulla ethmoidalis should be opened, and very cautiously

the lower portion or floors of the cells above the middle group should be excised or freely opened. To do this often requires several sittings, as it is much too dangerous a region to operate on when the parts are obscured by hæmorrhage. The use of supra-renal capsule extract, combined with cocaine, has the advantage of controlling hæmorrhage and rendering the parts anæsthetic.

In a few cases it is necessary to give a general anæsthetic, and for intra-nasal operations on the lower ethmoidal cells, nitrous oxide has certain advantages in that the position of the patient allows excellent illumination, while the anæsthesia is sufficiently prolonged to enable one to do as much as is safe or desirable at one sitting, without being followed by the disagreeable symptoms that attend chloroform anæsthesia.

Daily spraying with peroxide of hydrogen, which ferrets out

Nasal Accessory Sinus Disease.



FIG. 1.



FIG. 2.

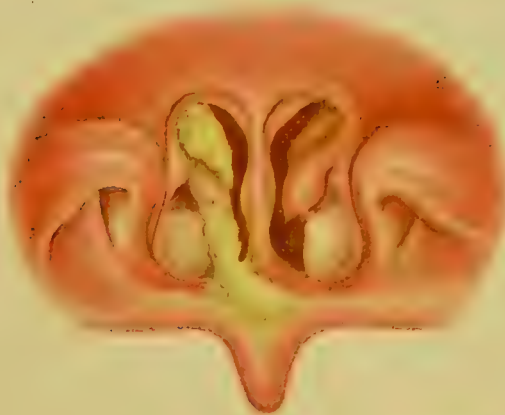


FIG. 3.



FIG. 4.



FIG. 5.

FIG. 1.—Case of right antral empyema. Pus is seen in the right middle meatus, the outer wall of which is swollen, giving the appearance of "cleft" or double middle turbinal on the right side.

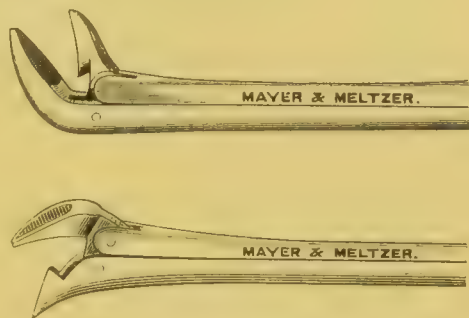
FIGS. 2 and 3.—Case of suppuration in the posterior ethmoidal cells on the right side. The pus appears above the middle turbinal, by both anterior and posterior rhinoscopy.

FIGS. 4 and 5.—Case of sphenoidal sinus suppuration. On anterior inspection some pus was seen far back near the floor of the nose. Posteriorly, the pus is seen after its escape from the sphenoidal sinus mainly on the right side. No pus is seen in the upper part of the nasal passages.

the pus in the honeycomb-like cavities, followed by other warm mild antiseptic douching should be persisted in, until all evidence of suppuration has ceased. Polypi and oedematous granulations may have to be removed at intervals, and perhaps further openings of the ethmoidal cells made from time to time. I have generally succeeded in obtaining a cure by these means.

If the upper cells are definitely implicated, and especially if the symptoms point to frontal sinus, orbital or intracranial complications, the external operation should be resorted to.

The posterior ethmoidal cells may be opened cautiously by a dental drill or cutting forceps, but in order to gain access to these cells it is necessary to remove the posterior portion of the middle turbinal body. Even then I have found the greatest difficulty in effectually dealing with these, the most inaccessible of all the nasal cavities; but the cutting forceps (*Fig.*



FIGS. 195 and 196.

FIG. 195.—The Author's Ethmoidal-cell Forceps, with pointed extremities, which readily pierce the softened and diseased bony structures.

FIG. 196.—The Author's Sphenoidal Sinus Cutting Forceps.

195), already alluded to, have proved of the greatest service in dealing with these difficult cases.

External Operations.—The fronto-ethmoidal cells are usually implicated in frontal sinus empyema, and even when the diagnosis, as is often the case, fails to distinguish between the two, they may be attacked by the Ogston-Luc operation, which has already been described. Entry through the inferior wall of the frontal sinus is often to be preferred, as it gives better opportunities of dealing with both the anterior and posterior ethmoidal cells. By turning forward or resecting sub-periosteally a portion of the lachrymal bone, the ethmoidal cells here are laid bare.

Others prefer gaining more complete access to the nasal cavity by splitting the nose and turning the parts aside, or by some operation such as Gussenbauer's and Ollier's, Fур-neaux Jordan's, etc.

Sub-periosteal removal of the anterior wall of the frontal sinus with temporary resection of the nasal bone (Killian's method), has its advocates, and Winckler subsequently removes the entire nasal portion of

the inferior wall of the frontal sinus so as to expose the ethmoid, which he cleans out, if necessary, to the lateral wall of the roof (cribriform plate). Then, after removal of the middle turbinated body, both the posterior cells and the sphenoidal sinus may be reached. But if the upper accessory cavities on both sides are involved, some such procedure as Gussenbauer's or Ollier's operation is preferable. These severe operations ought only to be performed when simpler methods have failed, or when severe and dangerous complications in the orbital or cranial cavities are threatening.

SPHENOIDAL SINUSITIS.

Suppurative catarrh of the sphenoidal sinus is a rare affection, and only a few undoubted cases have occurred in my own practice. When associated with caries of the body of the sphenoid it is probably due generally, though not necessarily, to syphilis or tuberculous disease.

Symptoms.—If the pus escapes, indefinite central headache, with mental torpor and depression, vertigo, cacosmia, and obscurity of vision are the usual symptoms complained of. The headache may be rather a sense of bursting in the centre of the head, or may be occipital, frontal, or temporal, and either central or unilateral or bilateral, whether, as is usually the case, one, or as more rarely happens, both sinuses are involved.

Dragging pain at the back of the eyeball is a frequent complaint. Catarrh in the Eustachian tube is a usual complication with tinnitus. The pus is usually discharged into the pharynx and comes away most readily on rising in the morning, or on stooping. When the sphenoidal ostium is occluded the bursting headache and neuralgic pains, the vertigo and mental depression are very marked symptoms, together with febrile disturbance, rigors, etc.

When the walls of the sinus yield to the pressure from within, blindness from pressure upwards involving the optic chiasma and nerves, or from lateral distension, compression of the motor nerves to the eye may cause ptosis and strabismus, and if amblyopia is not produced the visual fields are generally contracted. From compression or irritation of Meckel's ganglion, neuralgia of the fifth nerve may be severe. Further complications of the fifth nerve may occur, viz., suppurative meningitis, sub-dural or brain abscess, erosion of the carotid and other vessels, thrombosis in the cavernous sinus, and the usual symptoms that accompany these conditions. Schech reports a case

in which in addition there was excessive polyuria with a large amount of sugar. Mucous polypi may grow from the anterior wall of the sinus or in the superior meatus posteriorly, and the usual changes in the lining mucosa, fungous granulations and polypi, and also rarefying osteitis, etc., are found in the cavities in old-standing cases.

The pus escaping from the sphenoidal ostium runs into the fossæ of Rosenmüller and over the Eustachian orifice into the pharynx (see *Plate XXII*, *Figs. 4 and 5*). Some of the pus usually finds its way into the olfactory fissure and thence into the nasal passage; the quantity of discharge is often considerable in amount. Though it mainly passes down the posterior wall of the pharynx, a good deal may descend to the nasal passages, when it is blown out by the patient. Thus one of my patients affirmed that the pus came mostly from the front of the nose; it is therefore necessary to observe for oneself the location of the flow of pus and the direction it takes. By anterior rhinoscopy, pus is generally seen far back in the olfactory fissure, and only very rarely, when the nasal mucous membrane is very much atrophied, can the pus be seen flowing from the sphenoidal ostium.

Lewis reports a case in which, after thoroughly cleansing the nose, he was able to get an almost uninterrupted view of the anterior surface of the sphenoid, and he could detect a yellow spot of pus above its centre, which admitted a probe.

By posterior rhinoscopy the pus is seen in the naso-pharynx, especially in the fossæ of Rosenmüller, and often it may be observed in the nasal passages lying above the middle turbinal.

But the purulent discharge and the symptoms described may be due equally to disease of the posterior ethmoidal cells and the sphenoidal sinuses.

The only way to diagnose sphenoidal empyema with certainty is to pass a cannula into the sinus and draw off pus; the method of performing this will be described below in discussing the treatment.

It is well here to again point out that the sphenoidal sinuses may have no aperture communicating directly with the rhino-pharynx, as in a considerable percentage of subjects they open into the posterior ethmoidal cells.

The posterior ethmoidal cells and the sphenoidal sinuses, I have found, generally vary inversely in their degree of development—when the one is large, the others are small, and *vice versa*, the amount of pus discharged is therefore no guide to the location of its source. Ethmoidal cell disease can often only be excluded by the effect of treatment of the sphenoid sinuses.

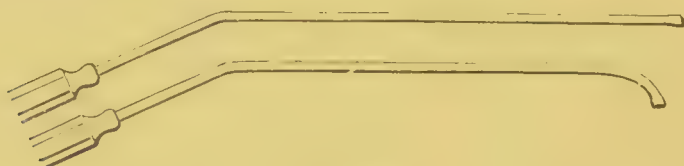


FIG. 117.

Lichtwitz's Cannulae for irrigation of the sphenoidal sinuses.

Treatment.—The only effectual treatment is to irrigate the sphenoidal sinus, and although it is a somewhat delicate procedure, it is by no means so difficult as it would at first sight appear. In a very few cases it is possible to introduce a fine cannula, e.g., Lichtwitz's, through the natural orifice, guiding the instrument upwards and backwards across the middle of the middle turbinal (in the line of the numbers 16, 11, 12, 7, in *Plate II*), till the inferior surface of the body of the sphenoid is felt, and then by directing the point to the upper and outer corner. After several gentle prods in different points in the area, the point of the cannula may be felt to enter the cavity. But apart from the usual difficulty of entering through the ostium, the disadvantages for drainage purposes of an orifice already more or less occluded by disease situated so much above the floor render it advisable to make an artificial opening, and for this we have two possible routes available: (a,) through the floor; (b,) through the anterior surface:—

(a.) *Entry through the inferior wall of the sinus.*—The floor of the sinus or the roof of the rhino-pharynx may be pierced by a drill introduced through the mouth. The operation is both more difficult and more dangerous than the second method, and has but few advocates. I mention it rather as a possibility than as an operation that can be recommended.

(b.) *Entry through the anterior surface* was originally suggested by Zuckerkandl, and first performed by Schäffer with a sharp gouge.

Before making an opening for drainage, the presence of pus should be rendered certain by passing a small trochar and cannula into the cavity and drawing off the contents by aspiration.

The plan I adopt is as follows :—

Having applied cocaine and supra-renal extract to the nasal passages, so as to render the mucosa insensitive and ischæmic, the cannula, with the trochar point just within its lumen, so as not to project at all, is introduced along the inferior meatus or along the lower border of the middle turbinal into the naso-pharynx. Then, by pressing the point upwards, the inferior surface is felt, and by gently withdrawing it a short distance the lower border of the anterior surface is found. Then by depressing the proximal end, the distal end is lifted still in contact with the anterior surface, till it has passed up about a $\frac{1}{4}$ of an inch. The trochar point is then pushed out, so as to puncture the thin wall, and the cannula passes in. Then withdrawing the trochar, the cannula is passed in till it comes in contact with the posterior wall. In this way the size of the sinus is estimated, and the depth of the anterior and posterior walls from the anterior nares gauged—data of considerable value when the forceps, curette, or trephine is used later to enlarge the opening.

Owing to the oblique direction in which the sinus is entered, the distance between the anterior and posterior walls is less than the greatest length of the sinuses. In a number of cadavers I found the cannula passed in for a distance varying between two-thirds to one inch, and in one patient it entered nine-sixteenths of an inch, in another fifteen-sixteenths. The distance from the centre of the nares to the anterior wall varied between $2\frac{1}{2}$ and $3\frac{1}{4}$ inches, and to the posterior wall between $2\frac{3}{4}$ and $3\frac{1}{8}$ inches. Myles puts the distance from the nares to the anterior wall of the sinus at $2\frac{7}{8}$ to $3''6$, and to the posterior wall $4''$. Wright, in eight cases, found the measurements were $2\frac{1}{2}$ to $3''$, and $3\frac{1}{2}''$ respectively, while Gleitzmann and Grünwald find the distance to the posterior wall varies between $3\frac{1}{2}$ and 4 inches. The distance to the natural ostium would be about $\frac{1}{4}$ -inch more than to the lower part of the anterior surface.

The cannula being *in situ*, suction is made by a syringe or other means, and if pus appears the diagnosis is confirmed. If no pus comes away, some warm normal saline solution may be introduced, and aspiration again applied, or some peroxide of hydrogen may be thrown in, when, if pus be present, it froths out through the free end of the cannula.

When pus has been proved to be present the anterior wall may be trephined or burred with a dental engine, or the opening may be enlarged in a downward direction by Hajek's hook, or better still by cutting forceps, such as the author's sphenoidal sinus forceps (*Fig. 196*).

The opening having been established, irrigations are made daily with some mild alkaline or antiseptic solution previously warmed.

With free drainage established, the sphenoidal sinus disease usually improves rapidly.

The dangers to be avoided in puncturing the cavity are perforation of the thin walls of the sinus, and injury to the internal branch of the spheno-palatine artery (see page 15). Gleitzmann has seen violent hæmorrhage follow an attempt to get an opening into the sphenoidal sinus. Myles has also seen severe hæmorrhage on attempting to remove part of the anterior wall, and Brewer and Bucklin a case of fatal hæmorrhage believed to be from the sphenoidal sinus following symptoms of quinsy. A consideration of the anatomical relations of the sinus will suffice to demonstrate the dangers of surgical interference unless most cautiously performed, for apart from the great variations in the size and development of the cavities and the thickness or thinness of the walls, there are often present the added dangers of softening of the thin walls due to the disease. Hence no one who is unaccustomed to deal with nasal affections would be justified in attempting these procedures, and every expert rhinologist would observe the utmost caution in their performance.

CHAPTER XIX.

*THROAT COMPLICATIONS OF
INFECTIOUS FEVERS, GOUT, RHEUMATISM, AND
SKIN COMPLAINTS.*

ENTERIC FEVER—SCARLATINA—MEASLES—SMALL-POX—CHICKEN-POX—
INFLUENZA—GOUT—RHEUMATISM—VARIOUS SKIN AFFECTIONS.

Introductory remarks.—The tissues of the upper respiratory tract are subject to much the same pathological conditions as similar tissues in other parts of the body, and many diseases of the skin are known to occur also in the mucous membrane of the nose and throat. The acute infectious fevers and most general infective diseases gain entrance through the air or food passages, and it is therefore natural that the portal of entry should often be directly implicated. The part played by the tonsil as a source of infection in diphtheria, tuberculosis and other diseases has already been fully discussed, but other infectious diseases may be ushered in or complicated by lesions in the throat and nose.

ENTERIC FEVER.

RHINAL AND PHARYNGEAL CATARRH, in greater or lesser degree, is frequent at the outset of typhoid fever, and, by extension to the Eustachian tube, may cause temporary deafness. EPISTAXIS, too, is fairly common at the commencement, especially during the first two or three days of fever; in fact, nose bleeding with fever and headache arising in a healthy adult should suggest the possibility of typhoid fever.

Erythema of the pharynx and fauces is not uncommon in the earlier stages, but though by no means a constant lesion, its occurrence is of no prognostic import. Occasionally the inflammation of the pharynx is attended with an herpetic eruption on

the mucous membrane; this is a somewhat painful condition, but it usually subsides spontaneously. Enlargement of the adenoid tissue in the pharynx, and of the tonsils, is stated by Bartholow to take place at the same time as the thickening and deposition in the Peyer's patches in the intestines, and occasionally white elevated patches are seen on the tonsils or posterior wall of the pharynx, which go on to ulceration or necrosis. Osler records a fatal case in which there was irregular ulceration in the posterior wall of the pharynx, 2 cm. by 5 cm. leading directly into the submucous tissue, an aggravated form of the pharyngeal ulcers described by Murchison as having a round, oval, or irregular outline, from two lines to three-quarters of an inch in diameter. Croupous pharyngitis may occur apart from true diphtheria, and is generally of the worst augury. Thus Morell Mackenzie, in his work, refers to six cases reported by Oulmont, five of which terminated in death, whilst Peter states that all the instances he has met with have proved fatal. On the other hand, a secondary diphtheritic deposit may occur during typhoid fever.

The chief laryngeal affections are: (1.) Laryngitis; (2.) Ulceration; (3.) Perichondritis; (4.) Paralysis.

Laryngitis in enteric fever occurs in two forms, the acute and the chronic.

Acute Laryngeal Complications in enteric fever are of more frequent occurrence than is generally supposed. No doubt in many cases the semi-comatose state of the patient explains the absence of subjective symptoms which would attract attention to the larynx. I have observed one case, referred to below, in which enteric fever commenced with the laryngeal affection, and two similar cases are recorded by Schuster. In such cases of laryngo-typhoid, the infection seems at first to be focussed in the larynx, and the nature of the disease may be overlooked until the general symptoms become obvious. As a rule, the laryngeal affection does not develop until the fifteenth or sixteenth day.

Wilks has pointed out that there is a great tendency for these inflammatory changes in the larynx to undergo ulceration, and Hoffmann observed ulceration in twenty-eight cases out of two hundred and fifty, while in two thousand autopsies at Munich they were noted in one hundred and seven cases (Osler). The

commonest sites of ulceration are the tips and edges of the epiglottis and the neighbourhood of the vocal processes.

As to the nature of these ulcerations, there is difference of opinion. Fagge, Murchison, Kanthack, Drysdale, and most authorities regard them as secondary lesions, and not in any sense primary or specific lesions of enteric fever. On the other hand, Mackenzie, Rokitansky, and others contend that they are specific typhoid ulcerations. Kanthack and Drysdale have shown that the majority of the ulcers are due to pyococci, but a certain number, especially of the deeper ulcers, are associated with infection by the typhoid bacillus. As this is a point of considerable clinical importance, I will briefly allude to three cases of enteric fever coming under my observation, which appear to support such an opinion.

Ernest S., age 20, was admitted to the Royal Infirmary on the sixth day of his illness. He became very delirious, and on or about the seventeenth day there was well-marked laryngitis and considerable bronchial catarrh, and he was continuously coughing and expectorating about the bed. He improved somewhat, but had a relapse, and on the twenty-ninth day of his illness he developed symptoms of acute laryngitis with dyspnœa. A steam bed relieved him, but in the evening he died suddenly, probably from the rapid occurrence of œdema. At the autopsy, in addition to the usual *post-mortem* appearances of *enterica*, the epiglottis and arytenoid folds were œdematous, while the ventricular bands were extensively ulcerated, the ulcers being purulent and sloughing. The right vocal cord had an ulcer on the *processus vocalis*.

Fourteen days after the death of this patient one of the nurses who had been attending him developed symptoms of enteric fever. She had not been in contact with any other case of enteric fever, and every precaution in dealing with the fecal evacuations had been rigidly observed. The disease ran a peculiarly virulent and fatal course.

But another patient in a different ward, the only one who was under my care, began to develop febrile symptoms about the same time as the nurse. He was a man, aged 38, who had been in the ward for some months for aortic aneurysm. The earliest symptoms of his attack of enteric fever were intense cephalalgia, and, a few days later, laryngitis and bronchitis. I found some

hyperæmia and superficial ulceration of the vocal cords about the eighth day, but he had had syphilitic laryngitis years before, and his larynx had been permanently damaged. He developed typical symptoms of enteric fever, but on the ninth day he became very cyanosed and collapsed from respiratory failure, not due to laryngeal obstruction, and died in a few hours. At the autopsy, in addition to all the typical lesions of enteric fever in the second week, there was superficial ulceration on the *processus vocalis* of each cord and on the anterior surface of the arytenoid cartilages.

Now the question arises as to the manner in which this patient contracted the disease, for he was in a separate ward, and one in which there was no other patient with enteric fever. He was rarely, if ever, visited by any friend from without, but I found that he had been in the habit of going to visit a patient in the ward where the delirious patient, Ernest S., was, although he never went within a few feet of E. S. I cannot avoid the conclusion that both my patient and his nurse contracted the disease from the expectoration of Ernest S., who was proved to have had typical typhoid lesions of the larynx while he was expectorating about the bed clothes.

Additional evidence of the correctness of this hypothesis was furnished by cultures of the Eberth-Gaffky bacillus in agar tubes inoculated from the ulcers on the arytenoids, and from the spleen.

These cases would, therefore, seem to explain the possible infectiousness of typhoid fever, as maintained by Budd, a view endorsed by Collicie, and brings home to us the necessity for more careful prophylaxis in cases exhibiting laryngeal complications. They also support the views of Landgraf and others who contend that the laryngeal ulcers of typhoid fever may be true typhoid ulcers.

The occurrence of ulcerations has also been attributed to mechanical causes acting on the inflamed and infiltrated mucosa. Dittrich, more especially, considers that they are the result of mechanical pressure of the larynx against the posterior pharyngeal wall, or arise from attrition.

(Edema of the larynx is a rare complication. Osler in recording one fatal case refers to the exhaustive article by Lünig,*

* Die Laryngo-und Tracheostenosen im Verlaufe des Abdominal typhus. *Arch. für klin. Chir.*, Band xxx.

wherein he states that œdema was present in nine out of a hundred and fifteen autopsies of enteric fever cases in which there were serious laryngeal complications.

Perichondritis with subsequent exfoliation of the cartilages is liable to follow ulceration, and the arytenoid cartilages or the cricoid may be denuded and become necrosed from this cause. A pseudo-diphtheritic pellicle may form on the epiglottis and within the larynx, sometimes requiring intubation.

The *chronic form*, according to Peter, does not commence until convalescence has been established; generally about two months after the attack of enteric fever has terminated. The voice becomes hoarse, there is a difficulty in phonation, and œdema is liable to supervene. This is an exceedingly grave complication, and very liable to terminate fatally; but even if the patient survives after tracheotomy, the condition is a very troublesome one to relieve, as cicatrices form and contract, producing a very persistent stenosis of the larynx necessitating retention of the cannula.

Paralysis of the vocal cords rarely results from enteric fever. Abductor or complete recurrent paralysis may occur on one or both sides, and may be due to pressure on the nerve by enlarged glands, or to toxic peripheral neuritis, or to nuclear disease, analogous to similar lesions of peripheral nerves or their central nuclei in other regions. Laryngeal paralysis is usually late in appearance during defervescence. Ankylosis from perichondritis is not very rare, but would be distinguished from paralysis by the thickening around the crico-arytenoid joint.

Prognosis.—The milder complication of catarrhal angina and laryngitis does not appear to modify the course of the disease any more than does a catarrhal bronchitis, but ulceration of the larynx is a very grave complication, the percentage of fatal cases being very high, while very few indeed of the diphtheritic cases survive. The prognosis of the chronic form of laryngeal ulceration is scarcely less grave, especially if œdema occurs. According to Sextier, of ten cases which necessitated tracheotomy, not one survived. Jobson Horne has found that in some cases dying from typhoid fever the laryngeal ulceration is tuberculous.

Treatment.—In the simple catarrhal class of cases, soothing inhalations or the sucking of ice, and drinking cool fluids will afford relief, while the fauces and pharynx may be sprayed with

a solution of permanganate of potash. When ulceration has occurred local antiseptic sprays or gargles should be used, and the teeth should be kept as clean as possible. For œdema, tracheotomy will have to be performed, but, if the patient survives, the resulting cicatricial stenosis will generally necessitate the tube being permanently retained.

The virulent type of enteric fever with which these lesions are associated generally precludes any active therapeutic measures directed to the larynx being successful.

SCARLATINA.

It is scarcely necessary to dwell at any length on the pharyngeal affections in scarlatina, since they constitute such a very essential feature in most cases of this fever, and are fully described in text books of general medicine.

As the result of an elaborate investigation conducted by Walter Dowson, of Bristol, on the rôle of the tonsil in scarlatinal infection, he is able to bring very strong evidence tending to prove that we should regard the tonsillar lesion characteristic of scarlet fever as the cause rather than the merely symptomatic consequence of a specific septicæmia, of the existence even of which we have no satisfactory evidence; that, in fact, as in diphtheria, the throat affection and cervical bubo of scarlet fever are the analogues of the chancre and inguinal bubo of syphilis, or of the intestinal ulcers and enlarged mesenteric glands of enteric fever.

The scarlatinal throat is generally a characteristic bright red colour localised in the fauces and tonsils, associated with a strawberry tongue and early rise of temperature. The early appearance of the rash usually confirms the diagnosis, but this is sometimes so slight and transient that a diagnosis has to be made from the appearance of the throat. Greater difficulty in determining the true nature of the throat condition occurs in patients who have already had scarlatina but who have been exposed again to infection. Dukes emphasises the danger of these cases of sore throat, "to all appearance often only simple acute tonsillitis, others having a membranous or sloughy appearance," inasmuch as without the appearance of any rash, they are capable of infecting others with scarlatina.

Scarlatina anginosa is the variety in which the tonsillar and

faucial inflammation is a marked feature. "The throat affection may be serious from the first; but more frequently, in a case which presents no very unusual features at the beginning, it undergoes aggravation either at the acme of the fever, or during the subsidence of the rash, or even after its disappearance . . . There may be abscess of the tonsil, or ulceration and gangrene, with œdema of the surrounding tissues; and supervening thereon, the glands in the neck may inflame and suppurate, and sinuses form" (Bristowe). With deep ulceration fatal hæmorrhage may occur. The inflammation in these anginose cases may extend to the larynx and the resulting infiltration and œdema produce aphonia, while painful deglutition occurs if the epiglottis is involved.

The larynx is seldom much affected, and œdema and ulceration here are much less frequent than in either measles or enteric fever, though all these lesions are met with at times.

A croupous laryngitis may arise by extension from the fauces—a very dangerous complication. Though diphtheria often follows scarlatina, it is only very rarely that a false membrane occurring in the course of the fever is truly diphtheritic.

PERICHONDRITIS of the laryngeal cartilages may occur and is very liable to suppurate and be followed by necrosis.

Later, and occurring as a complication of general anasarca, non-inflammatory œdema of the larynx may arise, and is liable to prove very rapidly fatal.

There is a great tendency for the pharyngeal lesions to spread by the Eustachian tube to the middle ear, but this complication is by no means limited to cases of scarlatina, and is very liable to supervene in measles and other exanthemata with marked pharyngitis. The accessory nasal sinuses are sometimes involved, and acute or chronic sinusitis may result.

Treatment.—In the less severe cases, simple soothing and antiseptic gargles and sprays are useful, while in the more aggravated throat affections, poultices or hot fomentations should be applied externally and the fauces swabbed out or sprayed with some powerful antiseptic, such as biniodide of mercury (1 in 2000), listerine, or sanitas (1 in 6). A tonic treatment and highly nourishing diet are called for in the anginose form of scarlatina.

The possible necessity for tracheotomy should always be borne in mind.

MEASLES.

(1.) A certain amount of catarrhal pharyngitis and laryngitis is present in almost every case of measles, but the severity of the throat complications varies greatly, being influenced considerably by family predisposition, and tends to be especially severe in some epidemics. The swelling of the laryngeal mucosa may produce great dyspnoea, as well as the usual hoarseness and cough.

(2.) In the stage of invasion, the so-called "endanthem," a mottled, punctate, star-shaped, or granular rash of the soft palate, fauces, tonsils, giving a stippled redness to the parts; and, according to Lori, of the laryngeal mucous membrane, followed by an eruption of small red points, may be a valuable diagnostic sign before the appearance of the characteristic cutaneous rash. Of greater value as an aid to early diagnosis prior to the appearance of the rash are Filatow's or Koplik's spots, small irregular spots of a bright red colour, with a minute, bluish-white speck in the centre of each spot. They appear on the buccal and labial mucous membrane. "These red spots with the accompanying specks of a bluish-white colour are absolutely pathognomonic of beginning measles, and when seen can be relied upon as the forerunner of the skin exanthem" (Koplik). The spots vary in number from two or three to countless numbers, are invariably discrete, and appear from twenty-four hours to three days or even five days before the characteristic exanthem, and begin to fade as the general eruption reaches its height (see *Plate XXIII, Fig. 1*).

(3.) Spasmodic laryngitis or false croup is not unusual in young children in the early stages.

(4.) Membranous laryngitis, apart from true diphtheria, may occur in measles, generally coming on late in the attack as the cutaneous rash declines. It is a very dangerous complication, of course, the prognosis and symptoms being similar to the idiopathic form. Dr. Samuel West observes that "this variety of croup seldom begins until the eruption of measles is on the decline, or the process of desquamation has commenced."

(5.) Laryngeal ulcers are occasionally met with on the vocal cords, ventricular bands, or posterior laryngeal wall (Gerhardt). Fränkel records instances of deep ulceration of the vocal cords and over the arytenoid cartilages, extending to the perichon-

drium and causing partial necrosis of the cartilages. They are due to pyogenic cocci invading the tissues.

(6.) Gangrenous laryngitis is rare, and is generally associated with gangrene of the mouth, in ill-nourished, unhealthy children.

(7.) H. Smith records three cases in which paralysis of the intrinsic muscles of the larynx occurred as a sequel to a mild form of measles. The paralysis set in a few days after the subsidence of the fever, and lasted from six to ten days. In each case laryngoscopic examination revealed total bilateral paralysis of the abductors and adductors of the vocal cords.

Treatment.—For the simple catarrhal and membranous laryngitis the treatment is the same as for the idiopathic forms (see p. 167 and p. 171). For gangrenous cases no local treatment appears of much use, but antiseptic sprays should be applied.

SMALL-POX.

The pharynx and larynx are inflamed to a slight extent in most cases. Rühle in fifty-four autopsies found the larynx affected in all. In many cases the vesicles appear on the fauces, and these have been observed in the larynx as discrete, well defined white spots on the epiglottis, arytenoids, and vocal cords, and in the trachea and bronchi. The symptoms of pustules in the larynx are those of laryngitis, and generally come on about the sixth day, and a few days later œdema may supervene, but Rühle observed that the essential feature of the laryngeal complication was a diphtheritic membrane far more often than pustules. The formation of a false membrane generally commences about the tenth day. Interstitial laryngeal hæmorrhage often complicates the hæmorrhagic variety of small-pox, while perichondritis and muscular paralyses are also liable to arise.

Treatment.—The treatment of these complications of small-pox is the same as for similar idiopathic affections of the larynx. Cicatricial stenosis is liable to follow extensive ulcerations occurring during the course of the fever.

CHICKEN-POX.

The characteristic vesicles surrounded by a well-marked areola sometimes occurs on the fauces, tonsils, etc. After a few days, superficial ulcerations form and heal spontaneously.

INFLUENZA.

An acute catarrh of the pharynx is an essential feature in most cases of influenza, and the larynx and bronchi are generally affected to a less extent. In a few cases the inflammatory infiltration is œdematous, or breaks down, or is attended with the formation of a false membrane. Shelley has described a vesicular eruption on the soft palate, the vesicles appearing like small well boiled sago-grains. The tonsils may be much inflamed and swollen. A notable feature of all the throat complications of influenza is their tendency to be followed by a persistent chronic congestion, and Cohen considers that there is undoubtedly a vaso-motor paresis of both the blood vessels and lymph vessels.

Œdema, hæmorrhage and abscess of the larynx may occur. As in the pharynx, the acute trouble is very liable to be followed by chronic laryngitis. By extension from the naso-pharynx the ear is often implicated in the acute catarrhal inflammation. Acute inflammation of the accessory sinuses of the nose is not uncommon, and a very large percentage of chronic empyemas of these cavities date from an attack of influenza.

Paralysis of the vocal cords is occasionally due to influenza, and paralysis of the soft palate has been noted.

Treatment.—The treatment of the throat affections of influenza does not differ from similar inflammatory conditions of the pharynx and larynx from other causes.

GOUTY AFFECTIONS OF THE THROAT.

The throat manifestations of gout may assume the acute or chronic form, and both gout and rheumatism are undoubtedly a very frequent cause of throat affections. Yet, common as rheumatic affections of the throat are, still more frequently do we encounter gouty disease of the throat. This is probably in part due to the fact that gout is a more chronic disease than rheumatism. Again, many of the slighter rheumatic affections undergo spontaneous cure, and the more acute are either regarded as a distinct disease, *e.g.*, tonsillitis, or are merged in polyarticular acute rheumatism, while, on the other hand, gout is a constitutional diathesis, and the symptoms persist. Moreover, the throat is very often affected when no definite evidence of gout is present.

It is interesting and instructive to note that Morell Mackenzie observed a patient who was suffering from acute pharyngitis when the symptoms suddenly disappeared, and an acute attack of gout developed in the great toe of his right foot, but after three days the gouty inflammation of the toe disappeared and acute hyperæmia of the pharynx supervened.

Symptoms.—In acute gouty pharyngitis and tonsillitis the patient generally complains much of the pain, especially on swallowing, the act of deglutition causing sharp pain radiating to the ears. Irritating cough with expectoration of a little sticky mucus or blood is common. The tonsil does not suppurate, but the intense redness and soreness may yield suddenly to an acute articular attack. Similarly, *painful laryngitis* is suggestive of gout. I have seen a case in which nocturnal laryngeal spasms occurred whenever an error in diet rendered the patient gouty. Some degree of hoarseness is usual, and in well marked or persistent gouty laryngitis the voice may be quite lost.

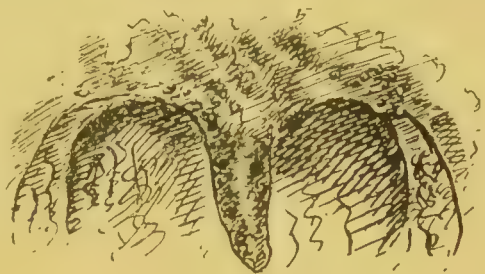


FIG. 198.
Gouty Pharyngitis.

In acute gouty pharyngitis, or laryngitis, the parts affected are acutely inflamed and bright red, the pharynx presenting an engorged, congested, glazed appearance. The inflammation, as a rule, is strikingly patchy in aspect. The uvula is sometimes much congested and swollen, or even œdematous. The tonsils do not suppurate, but the intense redness and soreness, as has been remarked above, may yield suddenly to an acute articular attack. The *gouty pharynx* is usually *excessively irritable*, and in many cases it is quite impossible to get a view of the larynx, even the most cautious and gentle attempts at laryngoscopy setting up such violent retching that one is compelled to desist, despite the application of cocaine

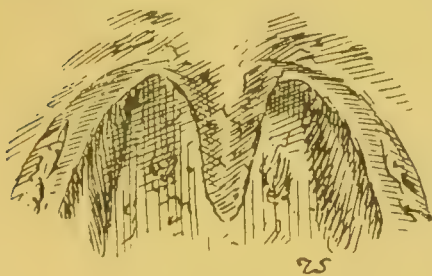


FIG. 199.

solution. Chronic disease of the pharynx generally results in considerable *thickening of the lateral walls*; indeed, such bilateral thickening, with a sense of uneasiness or pain of a darting character and shooting up to the ears, is strongly indicative of gout, and careful inquiry in the family and personal history should be made, while any evidence of the gouty diathesis in the general condition of the patient should be sought for.

Objectively the vocal cords will be found brightly injected; in slighter cases the hyperæmia is patchy; in the more severe, the redness of the cords is completely diffused, and the thickening, with irregularity of the margins, may be sufficient to produce aphonia. The ventricular bands and inter-arytenoid folds are usually similarly affected. Small tophi have been observed on a vocal cord and in the crico-arytenoid joint, though such conditions are exceedingly rare. De Mussy had a case of granular pharyngitis in which masses of concretion, consisting of carbonate and urate of lime, were discharged several times daily from the mucous follicles.

Morell Mackenzie reported four typical cases of gout in the throat, in all of which there were other proofs of their true gouty nature. These cases were (1,) an acute œdema of the uvula, disappearing upon sudden development of an ordinary attack of podagra; (2,) a chronic inflammation of the posterior pillars of the fauces; (3,) a gouty deposit around the crico-arytenoid joints on both sides, causing permanent dysphonia; (4,) a gouty inflammation producing fungous ulcerations of the left ventricular band, resembling cancer so strongly, objectively as well as in the subjective symptoms, that both Krishaber and M. Mackenzie had suspected it to be cancer, and discussed the possible necessity for extirpation of the larynx. This candid statement by such an experienced observer shows most clearly how difficult it may be to arrive at a correct diagnosis.

A form of *rhinitis sicca* is sometimes observed in gouty patients.

Treatment of these gouty manifestations is that of gout generally, the only local treatment necessary being some sedative spray or lozenge, such as Formulæ No. 46 and No. 8.

The following is a favourite prescription with me : —

R	Tinct. colchici sem.	-	-	-	-	-	-	℥ xx
	Sodii salicylat.	-	-	-	-	-	-	grs. xx
	Tinct. digit.	-	-	-	-	-	-	℥ iij
	Aq. dest. ad	-	-	-	-	-	-	℥ ss

To be taken in a tumblerful of Vichy water (Celestin), twice or three times daily.

Of course, suitable dietetic rules must be laid down and rigidly adhered to.

RHEUMATIC AFFECTIONS OF THE THROAT.

The very intimate pathological connection between acute lacunar tonsillitis, peritonsillitis and acute rheumatism is now widely recognised; but it is important to remember that a large proportion both of acute and chronic pharyngitis and laryngitis are of rheumatic origin, for success in their treatment will very much depend on a correct diagnosis. Pain, stiffness and inflammation of the fauces very frequently precede an attack of acute rheumatism, and either subside or are disregarded when the acute joint symptoms are manifested. In other cases the throat symptoms persist for days or weeks without further development, and not seldom recur regularly whenever the patient is exposed to cold or damp.

There is nothing peculiar in the appearance of rheumatic inflammations; but in the more acute cases of rheumatic pharyngitis, the soft palate, especially toward the free margin, the pillars of the fauces, the tonsils and posterior pharyngeal wall will be found somewhat swollen and heightened in colour, and in some cases the uvula is œdematous and distinctly swollen. The large majority of cases of acute follicular tonsillitis are rheumatic. The other lymphoid tissue aggregations in the throat at the base of the tongue and in the naso-pharynx are very frequently similarly implicated, and they may be the seat of acute inflammation when other neighbouring parts have escaped.

Personally I have never seen ulceration in the pharynx which I have recognised as rheumatic in origin, but Freudenthal states that he has repeatedly seen benign ulcerations of the pharynx which he is unable to interpret in any other way, except that they are due to rheumatism. The cases he relates from his own practice were associated with rheumatic symptoms and the ulcers healed rapidly under anti-rheumatic drugs.

Freudenthal refers to similar cases observed by Thorner and Heryng, and to another case of Westbrook's, where extensive ulcerations of the pharynx took place, due to rheumatism. He further remarks that in Heryng's cases and his own "we have always an ulcer which was one-sided, solitary, and which remained solitary, too. It had a typical form and size, appeared under the picture of a catarrhal angina, and healed without leaving a cicatrix. * * * Regarding the seat of these ulcers, I do not think we are as yet justified in speaking of certain parts which are predisposed to them. Heryng, whose so-called benign ulcerations of the pharynx I now accept as of rheumatic origin, found them at the anterior pillars; I saw them in four out of five cases on the pharyngeal wall, while Thorner considers the posterior pillars of the fauces (one case of Freudenthal's), the root of the tongue, etc., as the places mostly predisposed to rheumatic attacks." Freudenthal's patients who were treated with salol were cured in three to six days.

Laryngeal affections of rheumatic origin may be easily recognised as such, where associated with general symptoms of rheumatism or with rheumatic pharyngitis and tonsillitis. But if occurring independently it is very easy to misinterpret their true nature, and often enough a correct diagnosis is possible only by excluding all other causes. The laryngoscopic appearance of rheumatic laryngitis is, as I have already remarked, neither characteristic nor peculiar, but is merely that of simple laryngitis.

It appears, however, to have a tendency to affect the crico-arytenoid joints. Swelling may or may not be apparent, but if the joints are implicated, the movements of the corresponding vocal cords are impaired, and more or less persistent fixation of the vocal cords is liable to result. Doubtless some of the so-called rheumatic paralyses of the vocal cords are really due to crico-arytenoid ankylosis from rheumatic arthritis. Nevertheless, it seems impossible to account for other cases of true vocal cord paralysis except by rheumatism, and I certainly believe that rheumatic inflammation may rarely directly attack the intrinsic muscles of the larynx and peripheral nerves. In any case, the diagnosis of rheumatic paralysis of the vocal cord should never be made until all other possible organic causes of the paralysis have been excluded, and undoubtedly such cases of rheumatic paralysis are only of very rare occurrence.

Rheumatic affections of the nose are uncommon, but very painful swelling of the turbinates associated with acute articular rheumatism, and only relieved by anti-rheumatic treatment, has been noted by Freudenthal. Epistaxis sometimes occurs in patients undergoing treatment by salicylates.

Treatment.—The usual general treatment for rheumatic affections is called for, local applications by sprays, gargles or lozenges being mainly palliative. Guaiacum lozenges are useful in some of the subacute and chronic affections, and other local remedies are mentioned in the sections on pharyngitis, tonsillitis and laryngitis.

SKIN AFFECTIONS.

Many skin diseases have their counter part in the throat and nose besides lupus and leprosy, which are described fully elsewhere.

Urticaria of the respiratory passages may precede or follow the cutaneous eruption. Cough, dyspnoea and local irritation are the main symptoms. The local appearances resemble inflammatory swelling or œdema, and the glottic obstruction may be severe and even fatal.

Pemphigus appears in the pharynx or larynx in the form of vesicles, or "as a white fibrinous exudate which peels off, leaving the mucous membrane practically intact" (Bosworth).

Mycosis fungoides.—De Havilland Hall had a patient in whom, in addition to numerous tumours all over the body and limbs, tumours were seen on the posterior and lateral walls of the pharynx and on the left arytenoid cartilage.

Molluscum pendulum.—Furret has observed a small pedunculated tumour of this affection on the tonsil in association with similar tumours on the skin.

Actinomycosis may cause local red infiltration and brawny swelling with indented surface, and peculiar yellow points may sometimes be observed in the granulations. The affection is characterised by chronicity. The diagnosis depends on the detection of the *streptothrix actinomycotica*.

Foot and mouth disease (Aphtha epizootica).—Though strictly a disease of the lower animals, it is frequently communicated to man during epidemics of the complaint. It is an acute febrile disease characterised by the formation of a vesicular eruption

which is most usually situated on the mucous membrane of the mouth (tongue, cheeks and lips), and the skin of the fingers.

In some cases there is an offensive discharge from the nose.

Bussenius and Siegel have cultivated a small, ovoid, slightly blue bacillus from cases of foot and mouth disease and from children suffering from it ; they further produced the disease in healthy animals from inoculation of cultures of this bacillus.

M'Fadyean states that it is not improbable that some outbreaks of so-called "stomatitis epidemica" in mankind may turn out to be foot and mouth disease, and in all cases of such a character careful inquiry should be made into the possibility of the infection.

CHAPTER XX.

FOREIGN BODIES IN THE UPPER RESPIRATORY
AND ALIMENTARY TRACTS.

IT is convenient for clinical reasons to describe the symptoms and treatment of foreign bodies in the fauces, pharynx, rhinopharynx, larynx, œsophagus and trachea together, because in the first place, the power of localising sensations in the throat is very imperfect, the irritation from whatever part arising, being always subjectively referred to one common region, viz., the front part of the neck corresponding to the larynx and upper part of the trachea, *the laryngo-tracheal region*; and, secondly, because the foreign body is always liable to pass from one region to another, with or without any alteration in the resulting symptoms.

I.—FOREIGN BODIES IN THE THROAT, TRACHEA AND
ŒSOPHAGUS.

It is unnecessary to enumerate the many varieties of foreign bodies that are found in the throat, but it is usually only sharp-pointed bodies that become impacted in the fauces, more rounded bodies generally passing down as far as the larynx or into the bronchi.

Favourite sites for the location of sharp bodies are the tonsils, pillar of the fauces, lingual tonsil, or vallecula, and, if they pass further down, the pyriform sinuses, or else they may lie between the ventricles of Morgagni, or across the glottic aperture. In the œsophagus substances are apt to lodge opposite the cricoid cartilage, the lowest and narrowest portion of the pharynx, or else they may not be arrested until they have passed into the œsophagus or stomach.

Bony trabeculae and cartilaginous nodules in the tonsil and lateral wall of the pharynx have been found, and may be mistaken for foreign bodies. It is stated in recorded cases that they were instances of dislocated styloid process, but it seems more probable that the cartilaginous and osseous structures have a different origin (see page 105).

Symptoms.—The impaction of any sharp body in the tissues gives rise to pain and irritation, and when a body invades the larynx, laryngeal spasm and urgent dyspnoea are caused. But it should be remembered that often after the first few moments the larynx soon tolerates its presence, and the urgent symptoms subside, so that they may become so slight as to lead to a false impression that the difficulty is over, unless the invading body be large or so placed as to cause almost complete occlusion of the glottic aperture. Even when a coin lies right across the glottis, with its margins in the ventricles, the only subjective symptom after the initial glottic spasm, as long as the patient remains at rest, may be loss of voice. But when the glottic aperture is filled with the foreign body asphyxia occurs with rapid or even apparently sudden death, and instances of sudden death attributed to syncope have been proved *post mortem* to be due to the lodgment of a mass of food which had passed into the larynx either during swallowing or during vomiting. In children, too, the presence of a foreign body in the larynx may cause symptoms hardly distinguishable from croupous laryngitis.

Hæmorrhage is sometimes copious from the tearing of the tissues or puncture of vessels by sharp-pointed bodies.

Secondary complications, such as inflammation, perichondritis, or ulceration may arise, and may cause abscesses to form, and the pus may burrow in various directions, and when occurring in the region of the larynx or trachea may set up mediastinal abscess.

When the foreign substance is arrested in the œsophagus, pain, and difficulty in deglutition are usually the prominent symptoms. The obstruction in the œsophagus, if long continued, may lead to perforation of the wall of the œsophagus or to the formation of a pouch. When a bronchus has been invaded the primary symptoms are urgent dyspnoea and cough, but after a variable period the presence of the foreign substance is apt to cause secondary pneumonia and pulmonary abscess or bronchiectasis.

Diagnosis and Treatment.—The cases of impacted foreign body in the air and upper food passages fall into two groups: (*a*), those in which there is no history of a foreign body in the throat; (*b*), those in which the patient complains of having a fish-bone or other foreign body in the throat. Further, there are numerous cases in which the patient believes that a foreign body has stuck in the throat, but in which there either has never been a foreign body, or in which the foreign body has stuck in the passages and has been subsequently expectorated or has passed into the stomach.

The first group should be remembered, for when called to a case of sudden loss of consciousness one must never forget the possibility of its being due to asphyxia from the lodgment of food in the larynx either during swallowing or during vomiting, or as the sequel to an epileptic fit.

When a patient comes with a history of a foreign body having stuck in the throat, unless the case is so urgent that immediate relief is essential, two facts should be borne in mind, *firstly*, that the sensations of the patient are often misleading in localising its position, for all sources of irritation in the upper air and food passages are referred to the laryngo-tracheal region, and *secondly* that on no account should we incur the risk of displacing the invading body by palpation or the use of a probang until its position and nature have been determined.

The examination, especially if the substance is angular or sharply pointed, such, for instance, as a fish-bone or pin, should begin with a thorough and methodical inspection of all the parts with a bright light, beginning with the fauces, noting carefully the regions behind the pillars of the fauces, and the supra-tonsillar region, next the base of the tongue and the glosso-epiglottic fossæ, the larynx, the pyriform sinuses and the region behind the larynx. The inspection should extend to the trachea, and it is often possible to see as far down as the bifurcation; and finally the rhino-pharynx should be searched. If nothing can be observed, and it is known that the substance is soft or rounded, and that it is not likely to be harmful if pushed into the stomach, then an œsophageal probang may be passed in order that this may be brought about. But coins and any angular body should not be pushed down, as gastrostomy may become necessary as the result.

By means of the Röntgen rays it will be possible to locate the position of all substances which are impenetrable to the rays, and if stereoscopic skiagrams are available, the position and direction may be rendered very exact indeed.

Palpation is open to the objection that the finger may displace a foreign body and cause it to pass lower down, or even to enter the trachea or œsophagus or some inaccessible region; moreover, a sharply-pointed body like a fish-bone or pin may have pierced the faucial or lingual tonsil or other tissues, and only a small portion remain unburied, which the finger may push further in until perhaps the projecting portion becomes entirely buried, thereby rendering its discovery and removal very much more difficult.

When dyspnœa is urgent and fatal asphyxia is threatened, no methodical examination may be possible. the finger may then be quickly but cautiously introduced as far as the larynx, and thus it may be possible to displace a body which is occluding the rima glottidis, while carefully avoiding pushing it into the trachea. Should this manœuvre fail and the case is not too urgent, the patient should be inverted, and in this position two or three sharp blows upon the back of the chest may dislodge the foreign body from the trachea or bronchus, and cause it to pass out through the glottic opening. Should these means prove futile, tracheotomy should be performed, and if nevertheless dyspnœa is still urgent, the patient should once more be inverted, when the body may pass down to the larynx, and it may be possible to remove it through the tracheal opening.

In the less urgent cases the absence of symptoms should never lead us to allow a foreign body to remain impacted until all justifiable means for its removal have been attempted, for very serious and even fatal secondary complications, to which allusion has already been made, may subsequently arise. Nor should the failure to detect the foreign body lead one to assert that it has been dislodged, until by a very thorough examination we have positively excluded its presence. In many patients who come before us after having swallowed something which stuck in the throat, the substance has passed away leaving a strangely vivid after-sensation which sometimes persists for days; in other cases there has only been a local injury on swallowing some angular or hard substance which gave the impression of something

having stuck ; in yet others there is only some local inflammation or irritation due to various causes, the imagination of the patient leading to the presumption of some fish-bone or hair having stuck in the throat. In all these cases careful examination alone justifies the assertion that the feeling of something in the throat is only an after-sensation.

The removal of most bodies that have lodged in the throat or larynx may be accomplished by forceps. Coins and flat bodies may lie across the glottis, with the margins in the ventricles, as in the case I have illustrated. For removing foreign bodies in this position the author's forceps (see page 225) are specially adapted.

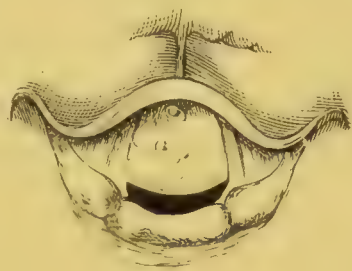


FIG. 200

If the substance has passed into the trachea or a bronchus (generally the right), and inversion before or after tracheotomy has failed to dislodge it, the tracheal wound should be kept open by means of threads in the margins of the tracheal wound, so that if subsequently the patient succeeds in coughing it up, it may with greater likelihood be expelled than it could be if a tracheotomy tube was worn. But it may be possible to pass long slender forceps down the trachea and even into the right or left bronchus, and the body extracted then and there. Coolidge, of Boston, succeeded in passing a urethroscope (half an inch in diameter and three inches long) through a tracheotomy incision down the trachea until he was able to see a foreign body which was lodged in the right bronchus, the upper end being half-an-inch below the bifurcation. It was seized and removed by alligator forceps. In passing the straight speculum into the trachea, the head and upper part of the body should be bent well backwards and to one side, as described by Schrötter. Coolidge's patient suffered no ill effects from the operation, and during the whole time respiration was carried on easily through the speculum, except for attempts at coughing just at first.

When a coin has passed into the oesophagus, its exact position should be determined by skiagraphy, and then its removal attempted by Sir T. Smith's coin-catcher, a method which has proved eminently successful by Mayo Robson in two cases. It

is simple in action and painless and should always be attempted before the final resort of lateral pharyngotomy. It might be possible to cause the ejection of a coin by apomorphine given hypodermically as an emetic.

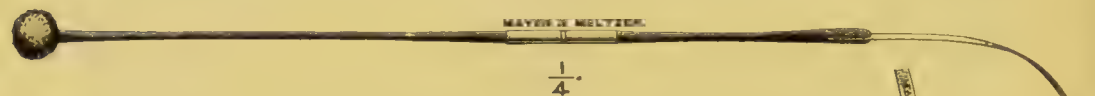


FIG. 201.

Smith's Esophageal Probang and Coin Catcher.



NAT. SIZE.

FIG. 202.

The Coin Catcher, natural size.

Schliep has found that the application of vinegar will soften fish-bones in fifteen or twenty minutes, while a 1 to 5 per cent. solution of hydrochloric acid, applied repeatedly by a cotton-wool tampon, will soften meat bones. If situated in the esophagus, small quantities of this solvent (1 per cent.) may be repeatedly swallowed.

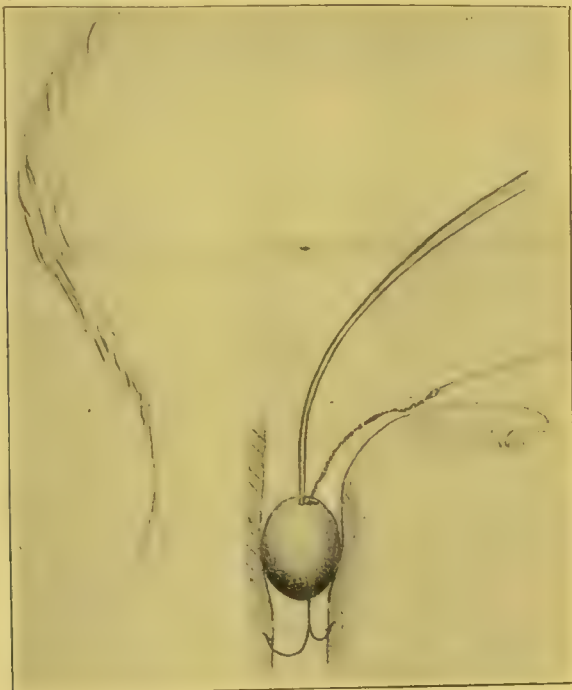


FIG. 203.

Christison's device for removing impacted barbed fish-hook from the esophagus, by boring a hole in an ivory ball and passing it down on the hook to cover the barbed extremities (WALKER).

If, on laryngoscopic examination, the body can be seen, it can generally be removed by suitable forceps, but the greatest care should be observed to avoid pushing it into the trachea.

It is often a very difficult matter to detect small bodies, such as pieces of fish-bone, which may get stuck in the lateral ary-epiglottic angle, being almost concealed from view on laryngoscopy, and it is well to bear in mind that the sensations of the patient are rarely any guide to the situation of a foreign body in the larynx.

An ingenious device of Sir Robert Christison succeeded in removing a barbed fish-hook which had stuck in a boy's œsophagus. The wire attached to it hung out of the boy's mouth. The drawing appended shows very well how this method was applied. A hole was driven through an ivory ball of a probang, not in its centre, as the divisions of the hook were unequal, and it was strung on to the wire, passed down to the barbs, which it covered, and then after first slightly pushing down the hook to disengage the barbs, it was easily extracted.

II.—FOREIGN BODIES IN THE NASAL PASSAGES. FOREIGN BODIES.

Various foreign bodies, such as small pebbles, buttons, beans pips, and pieces of slate pencil, are found at times in the nasal passages. Of course, if the patient is brought with the statement that some such body has been pushed into the nose, the diagnosis is easy enough, but very often months or years have elapsed, and meanwhile granulations may have sprung up from the irritated mucous membrane, concealing the foreign body, while the patient has completely forgotten having pushed it in, or it may have reached the nose in vomiting.

Vomited matter which has entered the nose, or a forgotten plug introduced to control epistaxis, may cause symptoms long after its introduction. Rarely, a tooth erupts into the nasal cavity and constitutes the foreign body.

The usual symptoms are pain in the nose and frontal region, with *unilateral* muco-purulent discharge, which is often fœtid and sometimes streaked with blood from ulceration or from the exuberant soft granulations that not infrequently conceal the body from inspection.

The conditions with which a foreign body may be confused are accessory sinus disease, malignant disease, osteoma, *ozæna*, lupus, tubercular disease, caries, and post-nasal adenoids.

Treatment consists in removal by means of forceps, a bent probe, or other means which may suggest themselves in any particular case. It is often necessary to employ some cleansing and antiseptic spray or douche after removal of the foreign body, and to treat the local inflammation which it may have caused.

PARASITES.

Various living creatures and fungi are found at times in the nasal passages, such as earwigs, centipedes, leeches, ascarides, oidium albicans, aspergillus, and they may give rise to considerable irritation and some rhinitis; but there is one affection which is particularly confined to tropical climates, in which the nasal passages are infested with maggots, the larvæ of the screw worm, *Sarcophaga Georgina* or *Lucilia Hominivora*, the affection being known by the name "Peenash."

"Peenash" usually attacks those who are affected with ozaena or catarrhal rhinitis. The symptoms are intense irritation of the nose, with agonising pain there and in the frontal region. There is profuse sanious muco-purulent discharge from the nose, with quantities of maggots escaping by the anterior and posterior nares, and sometimes from the ears. The subcutaneous tissues become inflamed and œdematous; generally symptoms of encephalitis supervene and the patient dies comatose. The prognosis is always grave.

The only treatment which offers good prospect of cure is the injection of pure chloroform into the nasal passages under the influence of an anæsthetic. If the maggots infest the nasal passages only, there is a prospect of cure; but when the accessory sinuses or the subcutaneous tissues were infiltrated, Kimball found that even 50 per cent. of carbolic acid, and 1 in 500 solution of perchloride of mercury, useless, while pure oil of turpentine only killed a few of the maggots. He advises the use of carbolised oil injections to relieve pain after the injection of pure chloroform. When the larvæ, or screw worms, or other parasites gain access to the accessory sinuses, the symptoms resemble those of sinusitis; in fact, acute or purulent sinusitis is liable to be set up. The treatment then usually involves opening into the affected sinus and injecting chloroform or some strong germicide.

APPENDIX.

FORMULÆ AND THERAPEUTIC METHODS—POST-MORTEM EXAMINATIONS.

FORMULÆ AND THERAPEUTIC METHODS.

INHALATIONS.

To be used with a Steam Inhaler.

1. R.—Compound Tincture of Benzoin 1 fl. dr., to a pint of Hot Water (140° F.) for each inhalation.
Sedative.
2. R.—Compound Tincture of Benzoin 1 fl. dr., with Chloroform 2 to 3 ℥. Mix and add to a pint of Hot water (140° F.).
Sedative in painful laryngitis.
3. R.—Creasote 80 ℥, light Magnesium Carbonate 30 grs., Distilled Water to 1 fl. oz. A teaspoonful to a pint of water for each inhalation.
Stimulant and antiseptic. Useful in ozæna and chronic laryngitis.
4. R.—Oil of Pine (Scotch or Swiss Pine) 40 ℥, light Magnes. Carb. 20 grs., Water to 1 fl. oz. Add to a pint of hot water and inhale.
Stimulant in chronic laryngitis.

PASTILS.

A good basis for Pastils is the following modified form of the Glyco-Gelatine Paste of the Throat Hospital, London:—

5. R.—Refined Gelatin 1 oz., Glycerin by weight 2½ ozs., Gum Acacia 2 drs., Orange Flower Water, or Triple Rose Water, 2 fl. ozs. Isinglass makes a pastil which dissolves more slowly.
6. R.—Menthol $\frac{1}{4}$ to $\frac{1}{2}$ gr., Cocaine Hydrochloride $\frac{1}{20}$ to $\frac{1}{8}$ gr., Glyco-Gelatin Paste q. s.
In irritable or painful laryngeal affections.
7. R.—Morphine Tartrate $\frac{1}{20}$ to $\frac{1}{40}$ gr., Emetine Hydrobromide or Hydrochloride $\frac{1}{60}$ gr., Glyco-Gelatin Paste q. s.
Sedative and expectorant.
8. R.—Powdered Cubebs gr. j, Cocaine Hydrochloride gr. $\frac{1}{20}$, Menthol gr. $\frac{1}{60}$, Glyco-Gelatin Paste q. s.
In sub-acute pharyngitis or laryngitis.

9. R.—Codeine $\frac{1}{4}$ to $\frac{1}{2}$ gr., Citric Acid $\frac{1}{2}$ gr., Elixir of Saccharin q. s., Glyco-Gelatin Paste q. s.
Very useful in allaying the irritable cough of phthisis.
10. R.—Emetine Hydrobromide $\frac{1}{60}$ gr., Tincture of Tolu 3 ℥, Codeine $\frac{1}{8}$ gr., Oil of Cubeb 1 ℥, Elixir of Saccharin q. s.
Forty minims of the Elixir of Saccharin will sweeten 8 ozs. of pastils.

GARGLES.

11. R.—Tincture of Phytolacca 4 fl. drs., Liquified Phenol 5 ℥, Diluted Acetic Acid 2 fl. drs., Tincture of Myrrh 1 fl. dr., Eau de Cologne 2 fl. drs., Water to 8 fl. ozs. (From Murrell's Handbook.)
Useful in catarrhal laryngitis and pharyngitis.
12. R.—Borax 40 grs., Tincture of Myrrh 30 ℥, Glycerin 1 fl. dr., Water to 4 fl. ozs.
13. R.—Potassium Chlorate 30 grs., Alum 30 grs., Sol. Morph. Hydrochl. 30 ℥, Diluted Hydrochloric Acid 60 ℥, Glycerin 4 fl. drs., Water to 4 fl. ozs.
14. R.—Tannic Acid 12 grs., Alcohol (90%) 6 ℥, Camphor Water to 1 fl. oz. (Th. H. Ph.)
Astringent.
15. R.—Gallic Acid 120 grs., Tannic Acid 360 grs. Water 1 fl. oz. Rub the acids, finely powdered, into a small quantity of water, then add water to one ounce.
For arresting hæmorrhage after excision of the tonsils.
The mixture may be slowly sipped if necessary. (Th. H. Ph.)

Goddard's Astringent Gargle.

16. R.—Red Rose Petals 2 drs., Diluted Sulphuric Acid $\frac{1}{2}$ fl. dr., Boiling Water 5 fl. ozs. Infuse and strain when cold, then add Clarified Honey 1 oz., Tannic Acid 40 grs., Alum 2 drs., Alcohol 90%, and Rose Water of each 6 fl. ozs.
17. R.—Solution of Hamamelis (B. P.) 1 fl. dr., Sodium Chloride 10 grs., Borax 5 grs., Distilled Water to 1 fl. oz.
18. R.—Phenazone 40 grs., Sodium Salicylate 60 grs., Potassium Chlorate 10 grs., Rose Water to 1 fl. oz.
Sedative and antiseptic in rheumatic sore throats.

MIXTURES.

19. R.—Apomorphine Hydrochloride $\frac{1}{20}$ gr., Syrup of Virginian Prune 30 ℥, Distilled Water to 1 fl. dr.
Every four hours.
20. R.—Ipecacuanha Wine 10 ℥, Tartarated Antimony $\frac{1}{50}$ gr., Cinnamon Water to 1 fl. dr.
For laryngitis and capillary bronchitis, given at frequent intervals.

21. R.—Liquid Extract of Myrtus Chekan 20 ℥
 " " " Yerba Santa 20 ℥
 " " " Grindelia Robusta 20 ℥
 " " " Quebracho 1 fl. dr.
 Brandy 2 fl. dr.

For spasmodic asthma, to be taken with half a tumblerful of hot water.

Warburton Begbie's Mixture (Edin. Royal Inf. Ph.).

22. R.—Diluted Hydrocyanic Acid $\frac{1}{2}$ fl. dr., Diluted Nitric Acid 3 fl. drs.
 Glycerin 1 fl. oz., Infusion of Quassia to 6 fl. ozs.
 A tablespoonful in a wineglassful of water three times daily.
 Sedative and tonic in phthisis.

INSUFFLATIONS.

Nasal.

23. R.—Bismuth Oxychloride $\frac{1}{2}$ gr., Morphine Acetate $\frac{1}{32}$ gr., Powdered Starch $\frac{1}{4}$ gr.
 Make one powder for insufflation.

Ferrier's Snuff.

24. R.—Morphine Hydrochloride 2 grs., Powd. Gum. Acacia 2 drs., Bismuth Subnitrate 6 drs.
 A small pinch to be insufflated at a time.
25. R.—Salicylic Acid (powd.) 10 grs., Tannic Acid (powd.) 60 grs., Bismuth Subcarbonate 60 grs.
 For nasal catarrh (Lefferts, New York).
26. R.—Iodoform in fine powder 3 grs., Morphine Tartrate $\frac{1}{2}$ to $\frac{1}{4}$ gr.
 One powder. Sedative and antiseptic in painful purulent conditions (Ed. Ryl. Inf. Ph.).

Menthol Snuff.

27. R.—Menthol 1 part, Boric Acid (powd.) 2 parts, Ammonium Chloride (powd.) 3 parts, Cocaine Hydrochloride $\frac{1}{100}$ part.
 Sedative and antiseptic.
28. R.—Borax (powder) 10 grs., Sodium Chloride 20 grs., Ammonium Chloride 10 grs., Camphor 1 gr.
 Mildly stimulating in chronic rhinitis.
29. R.—Citric Acid 60 grs., Sugar of Milk 30 grs., Powdered Gum Acacia 30 grs.
 Stimulating in atrophic rhinitis.

Laryngeal.

30. R.—Orthoform 20 grs., Menthol 2 grs., Powdered Dry Supra-Renal Capsule 10 grs., Ammonium Chloride 10 grs., Borax 10 grs., Powdered Tragacanth to 120 grs.

31. R.—Morphine Hydrochloride $\frac{1}{10}$ to $\frac{1}{4}$ gr., Bismuth Oxychloride to 3 grs.
For painful affections of the larynx, especially tubercular laryngitis.
32. R. Bismuth Subgallate in fine powder 1 gr., Boric Acid 1 gr., Morphine Hydrochloride $\frac{1}{12}$ to $\frac{1}{8}$ gr.
For tubercular ulcers.
33. R. Orthoform 60 grs., Amyloform 20 grs., Cocaine Hydrochloride 10 grs., Powdered Gum Acacia 30 grs.
Sedative and antiseptic in malignant ulceration.

SYRUPS.

Compound Syrup of Camphor (Bristol Royal Infir. Phar.).

34. R. Camphor 120 grs., Oil of Anise 2 fl. drs., Benzoic Acid 180 grs., Glacial Acetic Acid 7 fl. ozs., Tincture of Opium 11 fl. ozs., Squills 5 ozs., Ipecacuanha 2 ozs., Purified Sugar 28 lbs., Burnt Sugar enough to colour, Distilled Water to 4 gallons.
One teaspoonful to be taken occasionally.
(Approximately mij tincture of opium in 1 fl. dr.)

Linctus Limonis Compositus.

35. R. Morphine Hydrochloride $\frac{1}{2}$ gr., Diluted Hydrocyanic Acid 16 m , Spirit of Chloroform 12 m , Glycerin 2 fl. drs., Syrup of Lemons 2 fl. drs., Distilled Water to 1 fl. oz. One drachm contains $\frac{1}{16}$ gr of Morphine Hydrochloride.
One teaspoonful to be taken in water when necessary.

PAINTS.

36. R. Protargol 5 to 10 grs., Glycerin 1 fl. dr., Distilled Water 1 fl. dr.
Antiseptic in tonsillitis.
37. R.—Chromic Acid 45 grs., Distilled Water 1 fl. oz.

For application to ulcerated surfaces by means of a finely-pointed cotton-wool brush. To be followed quickly by an alkaline wash (Cent. Lond. Thr. Hosp.).

Pigments may be applied by means of a brush or by a cotton-wool carrier. The latter is more cleanly, clean cotton-wool being used each time. The carrier should be wetted, and applied to one end of a shred of cotton-wool, and then rotated in one direction until the whole shred of wool is firmly wound round it. The wool is then dipped in the solution and applied locally. In making *applications to the larynx* it is necessary firstly, to make certain that the wool has firmly gripped the holder so that it cannot possibly become detached and fall into the trachea; and, secondly, that the fluid applied will not drip from the wool when the latter is grasped by the vocal cords. Furthermore, to ensure its reaching the part of the larynx desired, one should use a laryngoscopic mirror held in the left hand (the patient himself holding out his tongue by a cloth in his right hand), while the carrier in the right hand is

guided to the larynx. The laryngeal spasm caused by local applications with the brush or cotton-wool holder is so intensely disagreeable and sometimes even alarming to a nervous patient that these methods should be avoided if possible (*see* Laryngeal Sprays). The previous use of cocaine in the form of sprays renders the laryngeal application more tolerable.

38. R. Dry Supra-Renal Capsule in powder 30 grs. Triturate for 5 minutes with $2\frac{1}{2}$ fl. drs. of Chloroform Water, filter through paper by aid of a pump; pass 15 ℥ Chloroform Water (or q. s. to make $2\frac{1}{2}$ fl. drs.) through the mare. To filtrate add Glycerin $1\frac{1}{2}$ fl. drs. and mix. (Approximately 10% solution.)

The use of supra-renal extract is liable to be followed by secondary hæmorrhage from cut vessels.

39. R.—Papain 4 grs., Lactic Acid 4 ℥, Distilled Water to 1 fl. oz.

40. R.—Silver Nitrate 10 to 60 grs., Distilled Water 1 fl. oz.

The previous application of a 10% solution of cocaine nitrate, which does not precipitate the silver solution, renders the application painless.

41. R.—Copper Sulphate 10 to 20 grs., Distilled Water 1 fl. oz.

42. R.—Zinc Chloride 10 to 30 grs., Hydrochloric Acid 1 to 3 ℥, Distilled Water to 1 fl. oz.

43. R.—Ferric Chloride (anhydrous) 30 to 120 grs., Distilled Water 1 fl. oz.

Mandl's Solution of Iodine (Schech).

44. R. Iodine $6\frac{1}{4}$ to 20 grs., Potassium Iodide 25 to 75 grs., Oil of Peppermint 3 ℥, Glycerin to 1 fl. oz.

FOR USE WITH THE OIL ATOMISER.



FIG. 204.

The "Aqual" Pocket Atomiser.

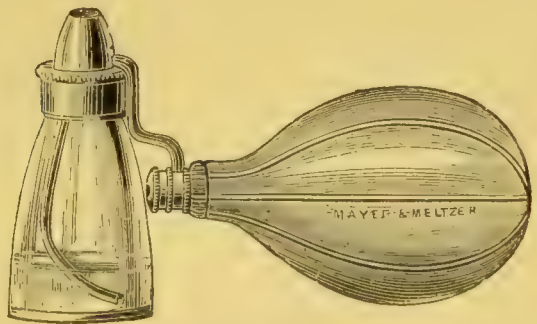


FIG. 205.

The Oil Atomiser.

45. R.—Eucalyptol 10 ℥, Terebene 10 ℥, Liquid Paraffin 1 fl. oz.
Antiseptic.

46. R.—Cocaine 5 grs., Menthol 15 grs., Terebene 10℥, Liquid Paraffin 1 fl. oz.
Sedative.
47. R. —Eucalyptol 10℥, Camphor 2 grs., Oil of Pine 8℥, Liquid Paraffin 1 fl. oz.
Stimulating in chronic rhinitis.
48. R. Eucalyptol 10℥, Aristol 10 grs., Menthol 20 grs., Liquid Paraffin 1 fl. oz.
In foetid rhinitis.
49. R. Eucalyptol 15℥, Menthol 10 grs., Terebene 10℥, Cocaine 6 grs., Liquid Paraffin 1 fl. oz.
In acute rhinitis.

DOUCHES AND SPRAYS.

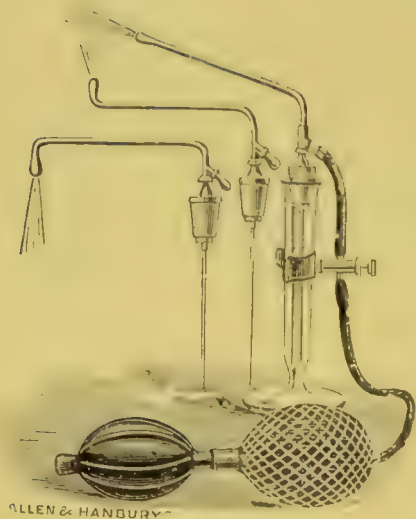


FIG. 206.

Convenient Sprays for nose, naso-pharynx, and larynx.

Fig. 206.—The rubber ball having been previously distended, the spray is started by compressing the button, and thus it can be worked with one hand.

Fig. 207.—Roger's "Aqual" Spray, with movable tips, enabling the spray to be sent in any direction. The manner of using the spray in the left hand, leaving the right free for operative purposes, is here shown.

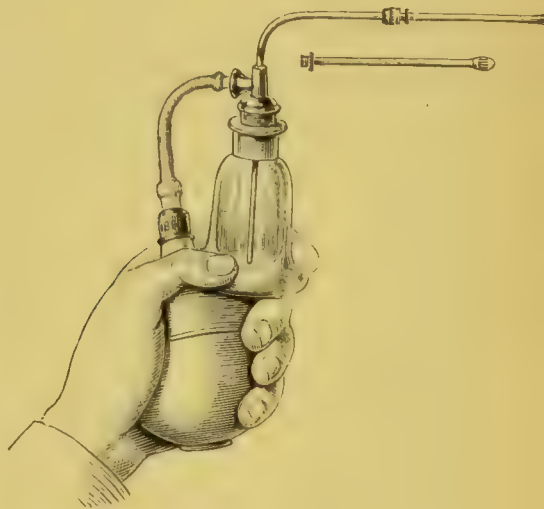


FIG. 207.

50. R.—Phenol 4 grs., Sodium Bicarbonate 15 grs., Borax 10 grs., Glycerin 45℥, Distilled Water to 1 fl. oz.
To be used with an equal quantity of warm water.

Spray up the nostrils with an atomiser or use with the nasal douche. Mildly detergent, useful in foetid discharge from the nose, and in hypertrophic rhinitis.

For applying fluids to the larynx it is desirable to use a fine spray during the act of phonation, and under the guidance of the laryngo-

scopic mirror. This avoids the intensely disagreeable laryngeal spasm so often set up by painting the larynx with a brush.

A fine spray is also a much pleasanter means of making applications to the nose, pharynx and rhino-pharynx than applications by the brush or cotton-wool swab.

51. R.—Listerine* 2 fl. drs., Borax 10 grs., Sodium Bicarbonate 10 grs., Water to 1 fl. oz.
Useful for removing crusts of secretion from the nose.
52. R.—Zinc Sulpho-Carbolate 2 grs., Distilled Water to 1 fl. oz.
Antiseptic.
53. R.—Papain 15 grs., Lactic Acid 15 ℥, Distilled Water to 1 fl. oz.
For dissolving diphtheritic membrane.
54. R.—Aluminium Aceto-Tartrate 1 oz., Distilled Water to 8 fl. ozs.
A tablespoonful to a pint of water to form a gargle, nasal douche or spray. Specially useful in ozæna (Edin. Ryl. Infirm. Pharm.).
55. R.—Sodium Salicylate 2½ drs., Borax 4 drs., Glycerin 4 fl. drs., Distilled Water to 8 fl. ozs.
Use 2 drs. to a pint of warm water.
56. R.—Cocaine Hydrochloride 30 grs., Morphine Hydrochloride 10 grs., Salicylic Acid ½ gr., Distilled Water to 1 fl. oz. (10% Cocaine).
Morphine is in some respects a physiological antidote to cocaine, tending to lessen its toxic action.
57. † R.—Cocaine Hydrochloride 15 grs., Morphine Hydrochloride 10 grs., Sodium Sulphate 6 grs., Distilled Water to 1 fl. oz.
Local anæsthetic.
58. R.—Hydrogen Peroxide (20 vol.) 1 fl. oz., Cocaine Hydrochloride 30 grs., Morphine Hydrochloride 10 grs.
A useful sedative and cleansing spray in tuberculous or malignant ulceration.
59. † R.—Eucaine Hydrochloride 15 grs., Sodium Sulphate 6 grs., Distilled Water to 1 fl. oz.
Local Anæsthetic.
60. R.—Equal parts of Formulæ 38 and 59 when local anæsthesia and ischæmia are desired while avoiding the use of cocaine.

* A mixture of the essential antiseptic constituents of thyme, eucalyptus, baptisia, gaultheria, and mentha arvensis in combination.

† The addition of sodium sulphate (2 per cent.) in Formulæ 57 and 59 is based on Wyatt Wingrave's investigations, which showed that the neutral sodium salt aided the absorption and effectiveness of the cocaine or eucaine; thus economising the alkaloid, and, in the case of cocaine, minimising the risk of constitutional symptoms.

NOTE ON POST-MORTEM EXAMINATIONS.

The Nose and the nasal accessory Sinuses.—The sphenoidal, posterior ethmoidal, and fronto-nasal sinuses may be laid freely open from above by removing with chisel and forceps the thin plate of bone forming the sella turcica, the olivary process, olfactory groove, and the portion of the orbital plate of the frontal bone in the floor of the anterior cerebral fossa lying just external to the cribriform plate, while the frontal sinus may be laid bare by removing its posterior wall and root external to the foramen cæcum. The upper part of the nasal fossæ may be inspected by removing the roof formed by the cribriform plate and crista galli. The maxillary antrum may be opened by raising the lip and cheek, incising the gingivo-buccal mucous membrane, then laying bare and opening into the wall of the antrum over the canine fossa. No facial deformity is involved by these examinations.

For a fuller inspection of the nasal cavities from in front the skin and subcutaneous tissues may be reflected from above downwards as far as the alæ nasi, drawing down the anterior half of the scalp after it has been divided as usual in opening the skull. The nasal bones should then be divided in the mid-line by a saw and turned to either side, carefully dividing, if necessary, the nasal processes of the superior maxillary bone. After examination is completed the nasal bones should be carefully brought together and the skin flap replaced. Very slight facial disfigurement is caused if this is carefully done.

The larynx and, if desired, the soft palate and faucial pillars may be removed, together with the tongue, by first dividing through the mouth the attachments of the tongue to the mucous membrane of the floor of the mouth and posteriorly to the cheeks, and cutting away the soft palate and pillars of the fauces from the hard palate and the buccal mucosa below it, and then passing a knife up from below (through the usual median incision in the neck for the removal of the trachea and œsophagus), so as to divide any remaining attachments of the larynx and tongue, the larynx and trachea being drawn down and held to one side or the other as convenience requires.

The larynx should be inspected carefully before it is opened in any way, and when it is desired to lay it open, it should not be divided, as is usually done, posteriorly through the interarytenoid fold, unless the posterior half of the larynx or trachea is free from morbid conditions for examination. Otherwise it should be opened by a longitudinal incision through the epiglottis. If it is desired to estimate slight alterations in the lumen of the larynx or trachea due to swellings or exudations, transverse sections, may be made after preliminary hardening by placing the specimen first in a 10 per cent. solution of nitric acid for twenty-four hours, and then, without washing, in Müller's fluid (Beuda).

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PLATES XXV—XXXVII.



TO ILLUSTRATE THE METHOD OF USING THE STEREOSCOPE.

Hold the Stereoscope *close* to the eyes so that both eyes look through the centres of the lenses, the picture being about twelve inches away. Three blurred images will be seen; then, fixing the eyes on the central image, gradually approach the picture until it appears clearly in focus and the several parts stand out in relief. It is essential that the Stereoscope should be perfectly parallel, that is to say, the Stereoscope must be held square with the picture, and if the image does not appear clear it is probably due to the Stereoscope not being held squarely. By slightly tilting it up or down the error will be corrected, and once the picture has been properly viewed all further difficulty will disappear.

An ordinary hand magnifying-glass will give an appearance of solidity and increase the effect of the plates, though not affording the realistic effect produced by the Stereoscope.

Preparation of a skull. The left maxilla bone has been sawn off, displaying the left maxillary antrum; the large orifice of communication with the nasal fossa is seen, and the partial subdivision of the cavity below by septa should be noted. The ethmoidal cells have been exposed by removing their outer walls; the extreme thinness of the bony partition dividing these cells from the orbital cavity will be observed. The frontal sinus of the right side has also been laid open.

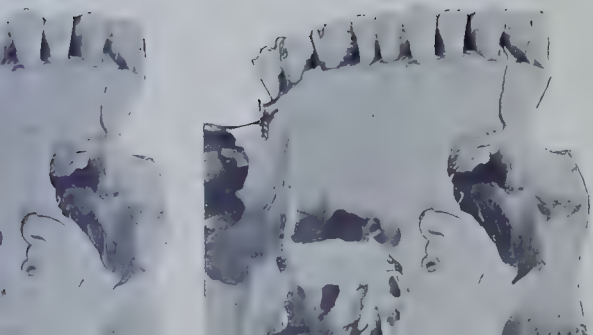


Fig. 2.—The same preparation as *Fig. 1*, but viewed from in front, so as to show the relations of the various sinuses and structures in the nose to the orbital cavity, etc., and very much as they would appear in anterior rhinoscopy. The right frontal sinus is laid open by removing the anterior wall, showing the incomplete bony septa which partially sub-divide the sinus longitudinally.



Fig. 2.

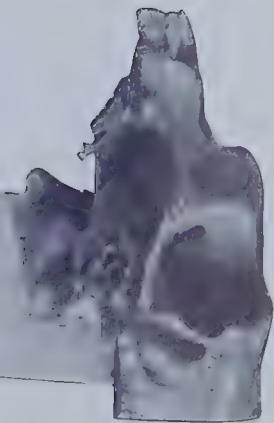


Fig. 2.—The same dissection as Fig. 1, but viewed from in front, so as to show the relations of the various structures in the nose to the eye and other parts of the face. Bristles have been placed in the apertures of the sphenoidal sinus and of one of the posterior ethmoidal cells. It will be noted that the *ostium maxillare* in this specimen is abnormally low, appearing below the border of the middle turbinated body, and not, as usual, in the hiatus semilunaris.

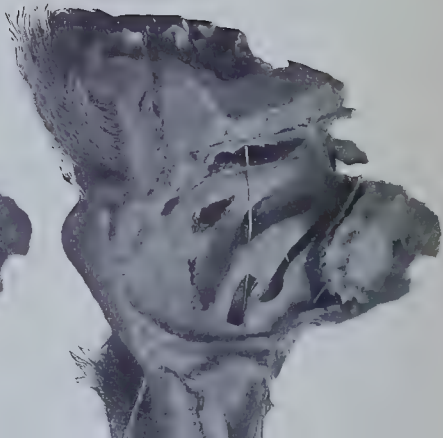
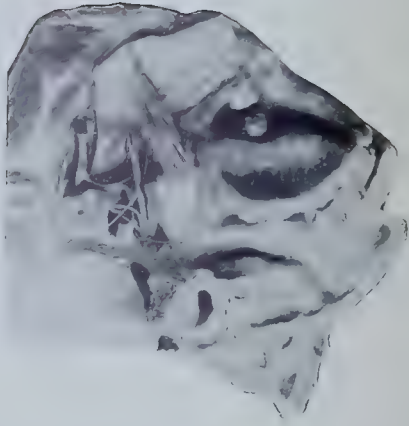


Fig. 2.

(2 and 3) the posterior portion of the maxillary sinus; (4) the posterior ethmoidal cells; (5) the well-marked transverse septum, the upward extension of this part of the antrum is displayed; (6) the middle turbinate body; (7) the inferior nasal septum; (8) the middle turbinate body.



Fig. 2.—Dissection showing the deep view of the right pterygoid region, with the branches of the third division of the fifth nerve, the otic ganglion, the Eustachian tube, the middle ear and its anatomical relations. (1) The middle turbinate body; (2) the inferior turbinate body; (3) the septum between the right and left sphenoidal sinuses; (4) the internal pterygoid muscle; (5) the inner surface of the membrana tympani; (6) canal for the tensor tympani muscle; (7) the geniculate ganglion of the facial nerve which is seen lying in the hiatus Fallopi; (8) canal for the Eustachian tube, separated from that for the tensor tympani above by the processes cochleariformis; (9) the otic ganglion; (10) inferior dental nerve; (11) lingual or gustatory nerve; (12) the external carotid artery dividing into petrosal and internal maxillary arteries; (13) two nerves, viz., above, the small petrosal joining the otic ganglion, and the branch to the tensor tympani muscle below; lower still, twigs from the plexus on (14) the middle meningeal artery form the sympathetic root to the ganglion; (16) branches to the tensor palati muscle; (17 and 18) nerve to the internal pterygoid muscle; (19) chorda tympani; (20 and 21) the auriculo-temporal nerve; (22) internal maxillary artery, from which ascends its middle meningeal branch.



backwards above the orbital cavity, the deviation of the median septum, the existence of other incomplete septa in each of the cavities, and on the left side the commencement of the fronto-nasal duct.



The same cartilages of the larynx are shown as in *Fig. 1*, but in addition, the cuneiform cartilages and the crico-thyroid membrane and true vocal cord. The cuneiform cartilages or cartilages of Wrisberg are seen lying in front of and extending above the arytenoid cartilages. The crico-thyroid membrane in its lateral portion is seen to end in the free, thickened, fibrous cord, the vocal cord. The superior cornu of the thyroid cartilage appears pointed instead of round, owing to an error in reproduction.

Fig. 2.—Lateral aspect.

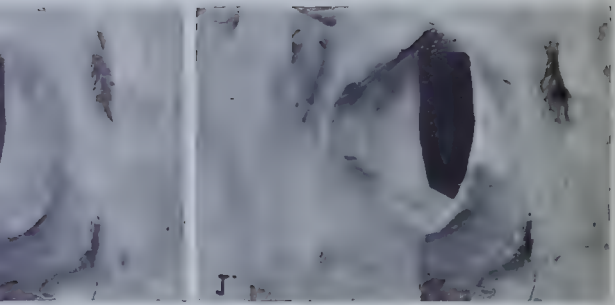


Fig. 2

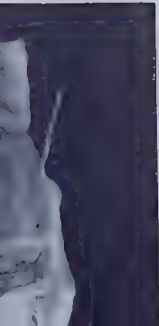
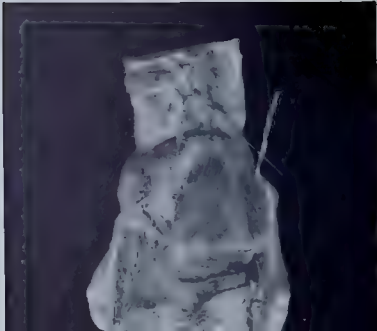


Fig. 1

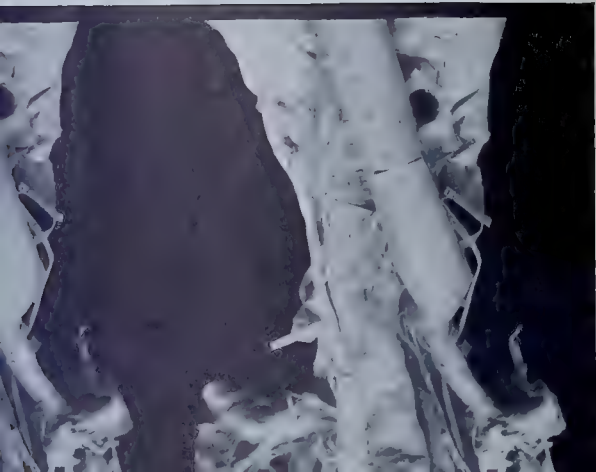
Fig. 2.—Laryngoscopic image. The same larynx, also life-size.

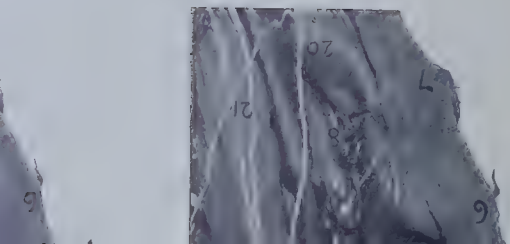


Dissection showing above the distribution of the superior laryngeal nerve to the mucous membrane of the larynx, which has been reflected. The twig communicating with the recurrent laryngeal is shown, and the recurrent laryngeal ascends from below, sending motor branches to the *posticus*, *lateralis*, and other intrinsic muscles.



recurrent branch, which winds backward around the arch and then ascends to the side of the trachea to the groove between the trachea and oesophagus, as on the right side. It is not seen in the dissection as the oesophagus has been drawn a little to the left to display the course of the right nerve in its ascent to the larynx, but its upward course to the larynx is similar on both sides. On the left side the superior laryngeal nerve is shown after its origin from the inferior ganglion of the vagus, passing inwards behind the carotid vessels to enter the larynx by piercing the thyro-hyoid membrane in conjunction with the superior laryngeal artery. The branches of the glossopharyngeal and the pharyngeal plexus, which supply sensory innervation to the pharyngeal constrictors, are likewise shown.





Section of the left temporal bone passing through the middle ear, and showing inner surface of the *membrana tympani*. (1) Head of the incus; (2) head of the malleus; (3) handle of the malleus; (4) tensor tympani muscle; (5) canal for the Eustachian tube; (6) attic; (7) chorda tympani; (8) the mastoid cells.



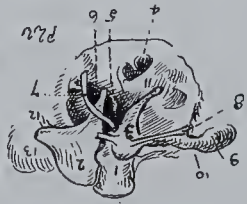


Fig. 2.—Right side.



Fig. 3.—Left side.

Figs. 2 and 3.—(1) Head of the malleus; (2) body of the incus; (3) handle of the malleus; (4) fenestra rotunda; (5) the stapes; (6) stapedius emerging from the pyramid towards the head of the stapes; (7) the chorda tympani; (8) anterior ligament of malleus; (9) osseus portion of Eustachian tube; (10) processes cochleariformis; (11) promontory; (12) posterior ligament of the incus; (13) mastoid antrum.

